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THE
SURGICAL CLINICS
OF
JOHN B. MURPHY, M.D.
AT
MERCY HOSPITAL, CHICAGO

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VOLUME II, 1913

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OPEN TREATMENT OF FRACTURES

An Address and Operation

BY MR. W. ARBUTHNOTT LANE, OF LONDON

At Dr. Murphy's Clinic, Saturday, November 23, 1912

DR. MURPHY: We are complimented and honored this morning by having with us one of the world's great surgeons, one of the men who has had the lime-light turned on him for many years, one of the men who is blazing the way in new fields, and one of the men who is suffering the vicissitudes that an explorer usually suffers. We know the criticism that has been heaped upon him for the especially prominent position he has taken in regard to bone surgery, and we know how well he has sustained himself under these trying ordeals. Gentlemen, I have the pleasure of presenting to you Mr. Lane, of London.

MR. LANE: Gentlemen: Dr. Murphy is always so gracious, always so kind, always so generous. Of course, he has exaggerated, and, if I might use the term, ridiculously so, the few little things I have done. He is so good, so magnificent in everything he undertakes, that he likes to see others who are his friends getting along in the same way.

I think it would be best for me to read my paper. There have come to us recently, and for the first time, an enormous number of cases that deal with these bone conditions. For years we have been fighting that in London, but it is only recently that the work has been taken up by the medical societies. We are overwhelmed with a terrible number of disasters resulting from improper care of fractures, and we feel ashamed. Every day we get letters from doctors in the country telling us of the failures in their work, and that they are being sued for malpractice because of the results. But I cannot be held responsible for the ignorance and uncleanliness of others.

One day my head assistant telephoned me that a doctor had just brought in a case with four inches of the femur gone. He said to me: "I told him that he must take the case back, that we did not want those kind of cases." I said, "No. Put him in the hospital and we will see what we can do." We put in an 18-inch plate, fastened it with large screws. That man is perfectly well. He has still three inches of shortening, but we could not help that. That is simply one of the many cases we get every day.

I will read to you what I have written, if you will permit.

GENTLEMEN: While I am fully conscious of the honor Dr. Murphy has done me in asking me to read a paper on the operative treatment of fractures before you, I quite recognize how difficult it is for me to say anything new on a subject which I seem to have worn threadbare long ago.

I was not led to resort to operative treatment from the observation of cases in the wards of the hospital where I found the surgeon had not only no knowledge whatever of the subsequent life-history of the patients he treated, but a ridiculously exaggerated or even quite an imaginary idea of the results of his treatment. I arrived at my conclusions from the study of the changes which the individual underwent in consequence of his leading a particular occupation. No subject can be more entrancing than the observation of the manner in which the skeleton alters with the mechanical relationship of the individual to his surroundings. Except for their comparative antiquity, I would refer you to certain papers * on the subject which illustrate evolution during a portion of the lifetime of the individual.

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* "The Causation, Pathology and Physiology of Several of the Deformities which Develop During Young Life," Guy's Hospital Reports, 1886.

"An Important Factor in the Causation of Some of the Curves which Develop in Mollities Ossium and Osteitis Deformans," Journal of Anatomy and Physiology, October, 1887.

"A Remarkable Manner in which Pressure Changes in the Skeleton may Reveal the Labor History of the Individual," Journal of Anatomy and Physiology, April, 1887.

"Can the Existence of a Tendency to Change in the Form of the Skeleton of the Parent Result in the Actuality of that Change in the Offspring?" Journal of Anatomy and Physiology, January, 1888.

"The Anatomy and Physiology of the Shoemaker," Journal of Anatomy and Physiology, July, 1888.

"The Result Produced Upon the Muscles, Bones, and Ligaments by the Habitual Exercise of Excessive Strain," British Medical Journal, December 1, 1888.

Not only will they help the reader to grasp the mechanics of the skeleton, but they will enable him to understand the forces which determine the changes in the drainage scheme in civilization producing intestinal stasis, a subject infinitely more interesting and important than that of fractures.

The knowledge of general principles acquired in this manner gave me confidence to ignore the storm of indignation that the operative treatment of simple fractures burst upon me, and which continued to beat uninterruptedly until the surgeons of America took an active interest in the subject and threw the weight of their opinion into the balance. How completely public opinion has changed in England, and with what abruptness, is shown by the report of the Committee on the treatment of fractures, which was appointed two years ago by the British Medical Association when I opened a debate on this subject, at which Dr. J. B. Murphy was present.

The Committee, which was a very strong one, was demanded by the opponents of the treatment, who little expected that it would verify up to the hilt everything I had claimed for it. That report will be published in the British Medical Journal, November 30, 1912.

REPORT OF THE COMMITTEE TO THE BRITISH MEDICAL ASSOCIATION ON THE TREATMENT OF FRACTURES

The Committee was appointed by the Council of the British Medical Association in response to a recommendation to that effect by the Section of Surgery at the annual meeting of the Association held in July, 1910. The reference from the Council to the Committee was—"To report on the ultimate results obtained in the treatment of simple fractures with or without operation." The following are the members of the Committee: Mr. W. J. Greer, Newport, Mon., *Chairman;* Mr. J. Grant Andrew, Glasgow; Mr. Charles W. Cathcart, Edinburgh; Mr. H. S. Crogg, London; Mr. Edward Deanesly, Wolverhampton; Mr. C. H. Fagge, London; Sir Victor Horsley, F.R.S., London;

Prof. J. Rutherford Morison, Newcastle-on-Tyne; Mr. Herbert J. Paterson, London; Mr. P. W. G. Sargent, London; Mr. W. Taylor, Dublin; Mr. Wilford Trotter, London; Mr. Richard Warren, London; with the following *ex officio*: Prof. Robert Saundby, M.D., Birmingham, *President of the Association;* Dr. Ewen J. Maclean, Cardiff, *Chairman of Representative Meetings;* Dr. J. A. Macdonald, LL.D., Taunton, *Chairman of Council;* Dr. Edwin Rayner, Stockport, *Treasurer*. The Committee was appointed in February, 1911, and held its first meeting in March, 1911.

Note.—The full report of the Committee is published in the British Medical Journal of November 30, 1912.

CONCLUSIONS

The following are the main conclusions deduced from an analysis and full consideration by the Committee of the material collected by the Committee's investigators:

I. The statistics relative to the non-operative treatment of fractures of the shafts of the long bones in children (under the age of fifteen years), with the exception of fractures of both bones of the forearm, show, as a rule, a high percentage of good results. These are unlikely to be improved upon materially by any other method of treatment. Operative results in children expressed in percentages are approximately the same as the non-operative. The relative figures are:

Non-operative Cases (cases, 1017), 90.5 per cent. good functional results.

Operative Cases (cases, 64), 93.6 per cent. good functional results.

II. It is possible, either by non-operative or by operative treatment, to obtain a high percentage of good results in children.

III. In comparison with the non-operative results in children, the aggregate results of non-operative treatment in those past childhood (*i. e.*, over the age of fifteen years) are not satisfactory.

IV. From the analysis of the age groups it is clear that there is a progressive depreciation of the functional result of non-operative treatment as age advances, that is to say, the older the patient, the worse the result.

V. In cases treated by immediate operation the deleterious influence of age upon the functional result is less marked.

VI. In nearly all age groups operative cases show a higher percentage of good results than non-operative cases.

VII. Although the functional result may be good with an indifferent anatomic result, the most certain way to obtain a good functional result is to secure a good anatomic result.

VIII. No method, whether non-operative or operative, which does not definitely promise a good anatomic result, should be accepted as the method of choice. For this reason mobilization and massage by themselves have not been found to secure a high percentage of good results. They are, however, valuable supplementary methods of treatment.

Similarly, of operative methods, those which secure reposition and absolute fixation of the fragments yield better results than methods which fall short of this; imperfect fixation of the fragments by wire or other suture has been found to be an unsatisfactory procedure in the treatment of fractures of the long bones, with the exception of the olecranon process of the ulna.

IX. Operative treatment should not be regarded as a method to be employed in consequence of the failure of non-operative measures, as the results of secondary operations compare very unfavorably with those of immediate operation.

In order to secure the most satisfactory results from operative treatment, it should be resorted to as soon after the accident as practicable.

X. It is necessary to insist that the operative treatment of fractures requires special skill and experience, and such facilities and surroundings as will insure asepsis. It is, therefore, not a method to be undertaken except by those who have constant practice and experience in such surgical procedures.

XI. A considerable proportion of the failures of operative treatment are due to infection of the wound, a possibility which may occur even with the best technic.

XII. The mortality directly due to the operative treatment of simple fractures of the long bones has been found to be so small that it cannot be urged as a sufficient reason against operative treatment.

XIII. For surgeons and practitioners who are unable to avail themselves of the operative method, the non-operative procedures are likely to remain for some time yet the more safe and serviceable.

When I started operating on simple fractures, I first made use of virgin silver wire to retain the fragments in accurate apposition, but about the end of 1894 I discarded it in favor of screws. I will quote a few lines from a paper * read before the Clinical Society of London on April 13, 1894, to illustrate my reasons for doing so.

"Now, as regards the best means of fixation of the fragments, in my earlier cases I used silver wire, but soon gave it up, as it was open to two great objections: First, as it is necessary to fasten its ends, it could only be passed in certain directions with safety, and to secure it satisfactorily one was at times obliged to incise the parts very freely. Secondly, one found that no amount of traction upon the ends of the wires would retain the surfaces of bone in accurate apposition after the grip of the lion forceps was relaxed. Therefore I decided to treat the bones as one would the broken leg of a table or chair. The surfaces were brought into accurate apposition and kept in their normal

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* "A Mode of Treating Simple Oblique Fractures of the Tibia and Fibula More Efficient Than Those in Common Use."

relationship by strong forceps. Holes were drilled above and below the forceps and screws were driven in. The screws could be passed in any direction, and they retained their surfaces in an apposition more accurate and more forcible than I fancy can be obtained by any other mechanical arrangement. As far as I know, they cause no subsequent trouble."

As regards my reasons for operating on simple fractures, I have nothing to add to what I stated plainly in that paper, namely, that—

"Operative measures offer to the patient the following advantages:

"(*a*) They at once relieve the patient from the pain of any movement of the fragments upon one another.

"(*b*) They free him from the tension and discomfort due to the extensive extravasation of blood between and into the tissues.

"(*c*) They shorten the duration of the period during which he is incapacitated from work, since union is practically by first intention, and, consequently, very rapid and perfect.

"(*d*) Lastly, and by far the most important, they leave his skeletal mechanics in the condition in which they were before he sustained the injury."

During the last twenty years I have operated upon a very large number of recent fractures at all ages, from early infancy to extreme old age, with the same uniform success. I have employed various methods at different times, endeavoring to devise means of fixation of the fragments in accurate apposition which shall be at the same time effectual and easy of application. I soon recognized that if the treatment by open operation was to become general, it was necessary that the procedure should be rendered as uncomplicated as possible.

The difficulty that has beset this treatment is that, if a foreign body is left in a wound, the surgeon must be infinitely more careful about asepsis than he is in the ordinary course of events, and careful in a degree proportionate to the bulk of the foreign body. It is because of insufficient aseptic precautions and to some extent from a want of familiarity with the use of wire, screws, etc., that we hear of so many complaints of the development of rarefying osteitis about a screw or wire perforating a bone, of screws working loose and consequently becoming ineffective for the purpose for which they were employed, and of the employment of such futile methods as ivory pegs, etc.

With the exercise of greater cleanliness and with an increasing manual dexterity these troubles will cease to arise. Why a surgeon should not be governed by the same principles by which an ordinary skilled mechanic works I cannot understand, but I presume that the surgeon has rarely had any training such as would fit him for this kind of work. The opposition to operative procedures which continued with such intensity for so long has suddenly diminished, because the more progressive surgeons have broken loose from tradition and have satisfied themselves that by operation alone can the greatest measures of success be obtained.

I do not propose to discuss the various methods I have adopted for retaining the fragments immovably in accurate apposition, but will confine myself to describing those which I now find most effective for the purpose.

In deciding as to whether the surgeon should operate upon any particular fracture, or whether he should be satisfied to treat the case by manipulation, splints, massage, active and passive movements, etc., the importance or not of restoring the fractured bone or bones to their normal form should be the guide. It is obvious that if there is no displacement of fragments, or if the displacement can be overcome by manipulation, etc., no operation is required in the ordinary case. Till surgeons generally have improved their technique sufficiently, and have acquired a greater familiarity with the operative treatment of fractures, a varying degree of risk must necessarily accompany any open operation, and this danger has to be taken into consideration. However, if the surgeon is careful and familiar with these operations, and his assistants are efficient and reliable, there is no risk in the operation, and, unless the bone is too thin, too soft, or too much comminuted, he ought to be able to obtain a perfect result anatomically and functionally. Alcohol plays a very important part in destroying the strength of the skeleton, by rendering the bones thin and fragile.

The surgeon must remember that the result of his treatment, whether operative or not, will probably be submitted to the test of radiography, and that a jury, instructed by counsel that our own creed teaches that the surgeon who has not placed the fragments in accurate apposition has not performed his work properly, are likely to regard any marked irregularity in the form of the bone as indicating a want of sufficient skill on the part of the surgeon, especially if such anatomic defect is associated with a marked loss of function. He must, therefore, be most careful in having fractures carefully *x*-rayed in planes crossing each other at right angles, so that any displacement may not escape his observation. A photograph in a single plane is not sufficient in the case of the long bones, however good it is. When a photograph cannot be obtained in several planes stereoscopic methods are very helpful. The details of the photograph should be carefully and clearly explained to the patient and to friends, and, whatever course of treatment is decided on, it would be advisable to obtain a written and witnessed acquiescence, so as to secure the medical attendant from subsequent loss, annoyance, and anxiety. To trust entirely to the now practically obsolete symptom called crepitus, and to an imaginary familiarity with the other clinical symptoms of fracture, is absurd in the present day, when every detail of any importance can be learnt by means of radiography, which is perfectly reliable in competent hands. In any case, the surgeon who does not insist on efficient radiograms being taken at the earliest moment in any suspicious case of injury renders himself liable to much criticism and financial loss in the future.

Perhaps the most awkward complications one may have to meet is fragility of the fragments, such as appears to exist to a great extent in alcoholics, and to a lesser degree in feeble children and in old age, and the softness of the bones in rickets. But this concerns the retention of the fragments in apposition rather than their reduction.

Delay in operating renders the replacement of the fragments more difficult, because the shortened soft parts or ties become rapidly less extensible and a correspondingly greater force is required to stretch them. This is a serious matter if the bones are fragile or comminuted. Again, the ends of the fractured bone and especially the distal fragments undergo a process of rarefaction which is very rapid and very marked in early life. The rate of rarefaction varies inversely as the age of the patient, but progresses out of all proportion in the first few days or weeks of life. This rarefaction means an insecure hold for the screws, and must be taken into serious consideration. The operation should be undertaken as soon as the skin can be effectually cleansed.

The only change in my technic in the last nineteen years has been the substitution, except in certain fractures, as those of the neck of the femur, of flat steel plates which are united to the proximal compact layer of the bone by screws threaded up to the head, which in the adult bone perforate and hold only the proximal layer of compact tissue. Their substitution for the longer screw has facilitated enormously the treatment of these fractured bones by operation, and has placed in the hands of the many a ready means of effecting a very important operative procedure with perfect safety, providing they will grasp the fact that unless an asepsis is employed which is much more elaborate than that required for operations in which no large foreign body is left in the wound, the screws will act as foreign bodies and will cause necrosis at the point of insertion, which will require their removal, and the end-result of the operation will probably be much worse than if the patient had been treated by what are euphoniously called conservative methods.

If the surgeon hopes to succeed in the treatment of simple fractures by operation, he must insure an extreme condition of asepsis of the wound by excluding the skin of the patient from the area of the wound, which should be of sufficient length to allow of free and easy access to the seat of fracture.

He should never touch the wound with his gloved hand, nor with any portion of an instrument that has touched his glove, since gloves may have pin-hole apertures in them and they are readily torn in the use of the heavy apparatus requisite for handling the fragments.

He should recognize that the expenditure of very little energy is requisite to bring the fragments of bone into accurate apposition if proper means be employed to do so, and that the accurate coaptation of fragments is brought about by the exercise of manual dexterity rather than by brute force.

In the case of fracture by direct injury the ends of the bone are protruded from the wound and are coaptated by powerful forceps, the limb being extended simultaneously.

When the fracture is oblique or spiral, the fragments are moved on one another by the rotation of very powerful gripping forceps, and their movement may be enormously facilitated by the rotation of an elevator placed between the fragments. This moves the fragments on one another with an irresistible force at the expense of a very little energy.

Not the least difficult nor the least important part of the operation is the closure of the wound. The edges of the skin incision must be brought accurately together and must be retained very securely in apposition, since any separation of the edges of the skin may result in infection of the wound. For this purpose Michel's clips are found to be the most effectual. When there is no tension they are introduced easily, but where there is much swelling and inflammation their fixation is a matter of the greatest difficulty. In such cases it can only be accomplished by securing accurate coaptation by means of specially constructed tenaculum forceps introduced closely together. The clips are then fixed near one another in the intervals between the forceps, and when they have all been attached the strain upon them is so evenly distributed that the forceps can be safely removed without any separation of the edges of the incision.

It would be well for a surgeon who has had no previous experience of the operative treatment of fractures to telescope the difficulties which may arise by seeing how these operations are done by observing the procedure adopted by an operator who has had special training and experience in this form of surgery.

To read, as one does, of surgeons who do not regard the development of sinuses, necrosis of bone, etc., as evidence of infection seems absurd at the present time. If the surgeon has the sense to recognize that these accidents are quite unjustifiable and are readily and certainly avoided with proper and efficient technic, he will soon cease to meet with them.

I cannot illustrate this latter better than by alluding to a paper* by Mr. Sampson in the Lancet, August 17, 1912, in which he shows that in not one of the 72 cases of simple fracture operated on by me at the Hospital for Sick Children was there the slightest evidence whatever of sepsis. He has spent much time and energy in the preparation of that paper and it will well repay perusal.

Where the surgeon will always experience anxiety is in operating on fractures of the upper extremity in the immediate vicinity of important nerve-trunks, and the situation of the incision and the mode of obtaining free access to the fragments may require much thought in devising it and skill in carrying it out.

Comminution of fragments may render an operation very difficult, and if associated with thinning and fragility of the compact bone, may put it beyond the pale of operative interference.

My experience in dealing with comminution when the density of the bone lends itself to operation is that it is usually wiser in the first instance to secure a sufficiently long steel plate to the lower fragment and then to connect the several loose fragments by small suitable plates to the lower piece. Later the lower fragment with the small pieces of bone secured to it and to one another can be fastened securely to the upper fragment by means of the long plate.

Do not hesitate to put plenty of metal into the wound, since it is exceedingly important that the fragments should be held together with sufficient security to permit of the movement of adjacent joints without endangering the junction in the least. This is particularly the case in the knee and in the joints of the foot and ankle, which readily stiffen if kept at rest for any time.

Also do not place a plate beneath the skin if it is possible to cover it up with muscle, but if it must be attached in this situation, take care that the skin incision is at some distance from it.

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* "The Operative Treatment of Simple Fracture of the Long Bones in Children," by H. H. Sampson.

If a plate placed subcutaneously causes any annoyance after union has taken place between the fragments, it is as well to remove it before the man resumes any arduous occupation.

In the case of fractures through or in the vicinity of an epiphyseal line if the epiphysis cannot be restored accurately to its normal position by manipulation the fracture should be freely exposed and perfect apposition effected. A plate or screw is hardly ever requisite in these fractures, but if by chance one has to be used, it should be removed immediately union has taken place, so as to avoid any control of the growing line. It is well for the surgeon to remember in this relationship a law which I devised, namely, that "the rate of bone formation in the several parts of a growing line varies inversely as the pressure." In no circumstances in any fracture should the surgeon be satisfied with anything other than the perfect restoration of the broken bone to its normal form. There must be no compromise in this class of surgery.

The class of fracture that makes most demand on the skill and ingenuity of the surgeon is the badly united fracture, and this difficulty is increased if two bones are involved, and especially if a long interval has elapsed since the receipt of the fracture and if the subject be a child. These operations are very much more difficult and are accompanied with more risk from hemorrhage than are recent fractures. The results obtained are also very rarely as good, because of the changes which develop in the joints whose functions have been affected by the faulty junction. I think that with better initial treatment such cases will soon cease to exist.

Whatever the extent of the shortening in badly united fractures, the surgeon should endeavor to correct it entirely or to reduce it as much as possible. There is, as far as I am aware, no limit to which the muscles, nerves, vessels, etc., can be stretched if only sufficient force can be exerted on the fragments. Such operations may make a very great demand on the skill and resources of the surgeon, and an increasing familiarity with them will enable him to obtain an increased measure of success.

All these difficulties are accentuated by comminution which may appear to make it impossible to establish continuity unless the limb be shortened very considerably, which in the case of the leg is not only a matter of physical importance, but affects very materially the esthetic aspect of the individual. This difficulty can be largely if not wholly overcome by the knowledge of a general law which I formulated, namely, that the skeleton represents the crystallization of lines of force which when exerted in one direction are laid down as compact tissue, when exerted in varying directions, as cancellous tissue.

Any alteration in a portion of the skeleton in the infant is followed by the formation of a new bone, the lines of force recrystallizing and the old bone outside the new lines of force is absorbed. The degree in which this takes place varies inversely with the age of the subject, and as age advances the crystallization, which is spoken of in surgical terms as callus, becomes limited more and more to the seat of fracture.

The same crystallization may be brought about late in life by engorging the part with blood. In this manner it is not necessary to bring the entire section of a shaft of a bone into accurate apposition, so sacrificing length of the limb, but if only a portion impacts the interval can be filled up by this means of overfeeding with blood.

In the case of the upper extremity this can be effected more readily by applying an elastic compress above the seat of fracture.

In the leg the same effect is best produced by attaching an apparatus by means of which the weight of the patient is transmitted from the tuber ischii to the splint beyond the foot.

Besides engorging the limb, such an apparatus enables the patient to walk about from an early period securing himself by two walking-sticks. You will find these modes of applying the general principle above alluded to most useful.

Perhaps the variety of recent fracture that causes the surgeons the greatest anxiety is the compound fracture, since he has to deal with the strong probability of infection. Personally, if I can obtain a reasonable approximation of fragments, I prefer to treat this form of fracture by splints or some similar fixation apparatus. If there were very definite sepsis, I would even prefer the fragments uniting in a bad position to accepting the risk of introducing screws into a septic wound.

If, however, the fracture is of such a nature that much deformity may result and there is room for its application, I employ a very long steel plate with many perforations, and after obtaining accurate apposition of fragments, I fix it by screws at a considerable distance from the seat of fracture, so that if any necrosis takes place about the screws, it does not interfere with the texture and nutrition of the bone in the immediate vicinity of the fracture.

Of course, one's treatment of this form of fracture must vary largely with the locality of the fracture and with the degree of infection. It is well to remember that organisms will remain quiescent for a long time in a fracture which has united firmly and apparently normally, and that the subsequent introduction of screws may stimulate them into active growth and so endanger a junction.

[Here Mr. Lane showed a number of lantern-slides illustrating various forms of fractures treated by him, and the subsequent results, making running remarks on each case.—Ed.]

Fracture of Left Tibia

HISTORY

Patient, male, aged twenty-four, comes to hospital on account of malunion of both bones of left leg.

Seven weeks ago the patient was standing with his back against a pile of timber, helping to unload iron beams. One of these beams, 15 feet long, rolled from the pile and pinned his left leg against the lumber pile. He was held there by this beam for fully half an hour before he was released. He was unable to walk, and noticed a false joint in the middle of the leg. He was taken to a hospital and the leg was placed in a short splint. In six weeks he was sent home by the doctor, who said he was well. His friends were dissatisfied and sent him to another hospital. There is some motion at the point of fracture and a malposition of fragments.

The skiagram shows that both the tibia and fibula were fractured, with a small interposing fragment between the two larger fragments. There appears to be a weak bony union of the fibula, with displacement of the fragments. The tibia has not united at all, and there is a malposition of the fragments (see skiagrams).

Dr. Murphy: While Mr. Lane is preparing for the operation I will show you some of the work that has been done here. [Showing pictures and plates.]

Case 1 (Figs. 1, 2, and 3).—This case was originally not recognized as a fracture. The patient was told that it was simply a sprain of the hip-joint. The man was permitted to walk about. The first skiagram was made three weeks after the injury was sustained. That picture was examined by four good men. Three said it was not a fractured hip-joint, and one insisted that it was. The man was permitted to go to Europe. It was really a case of impacted fracture of the femoral neck. The skiagram shows that very distinctly. If you look at it more carefully you will see that there is a little displacement of the under margin of the distal fragment. We exposed the fracture, freshened the surface which had become eburnated from friction, and nailed the detached head to the shortened neck with three wire nails. That man recovered completely from the operation, had a bony union, as shown in the skiagram, and was sent home. Four or five days after reaching home he had an embolus of the superior mesenteric artery from which he died. If you examine the last skiagram you will see that the head is firmly approximated to the neck of the bone, and that there is already evidence of bone regeneration. This skiagram was taken about six weeks after the operation. This shows also that the shaft end was alive; in fact, at the operation we found that the detached head was still alive, getting its nourishment through the ligamentum teres.

In the first skiagram (Fig. 1) it will be noted that there is but very little shortening of the neck of the femur and not the slightest deviation in the angle. Four months later there appears a considerable absorption of the neck, and finally two years and four

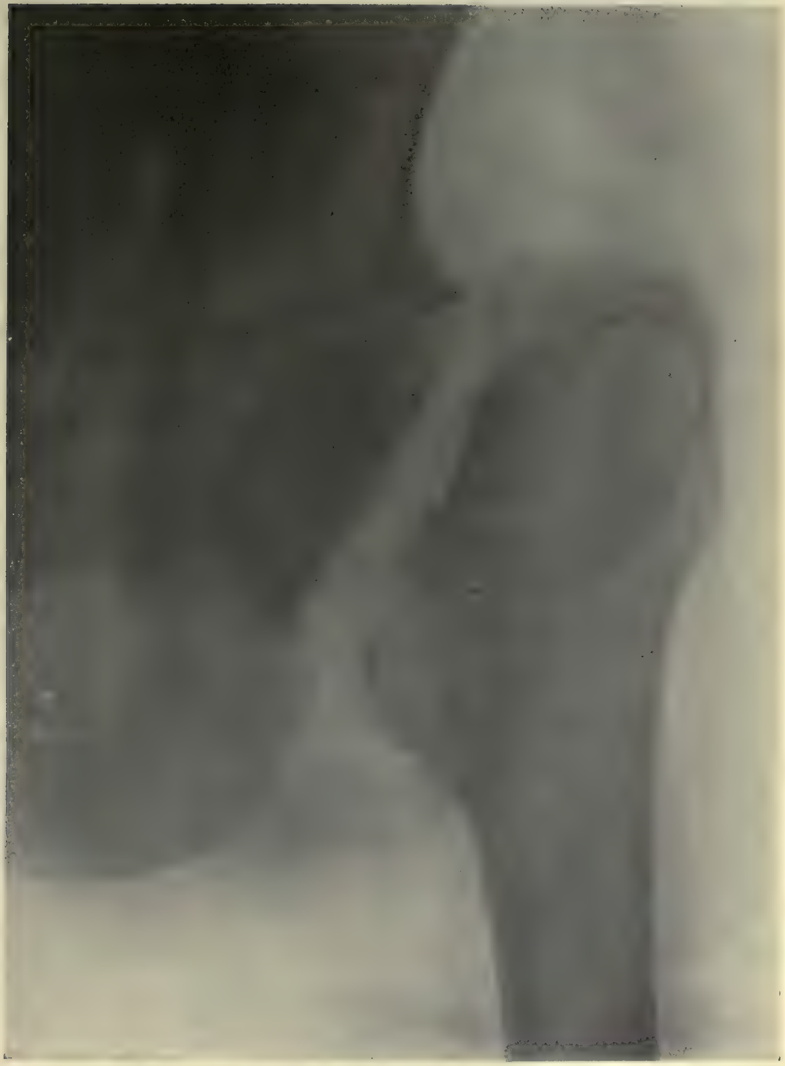


Fig. 1.—A fracture of the anatomic neck of the femur, with considerable absorption of bone and separation of the fragments. The head is in the acetabulum. The shaft is displaced upward slightly.

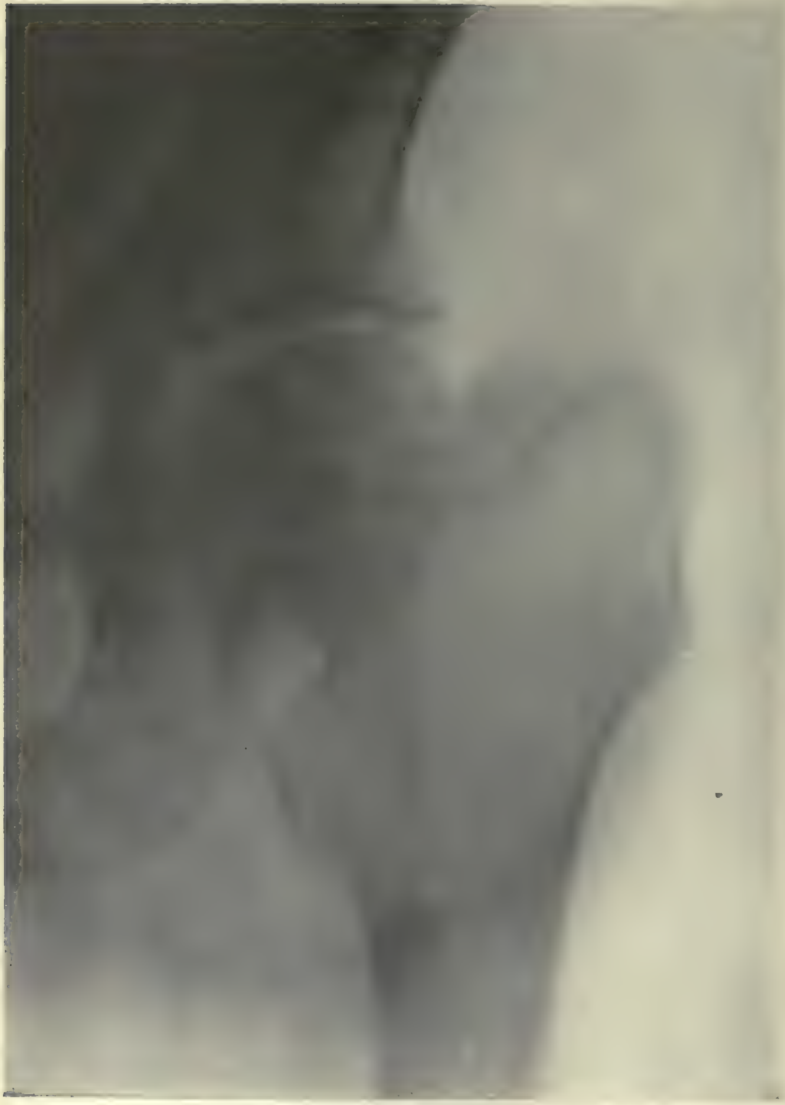


Fig. 2.—Another view of the same case taken from a different angle. Fracture not apparent, although it seems as though there had been a fracture with impaction of the fragments.

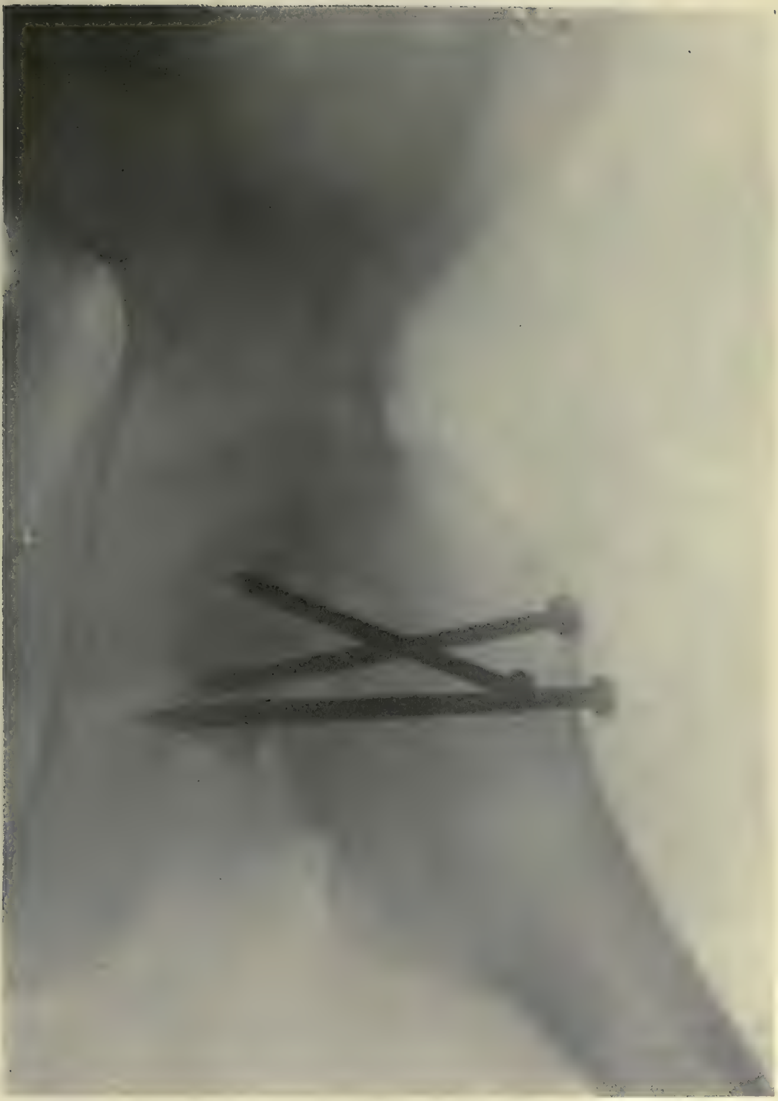


Fig. 3.—The fragments were exposed and the ends freshened. Approximation was secured and maintained by means of three wire nails driven through the shaft into the head. One of these nails penetrated the acetabulum and aided in maintaining fixation. Leg is abducted.

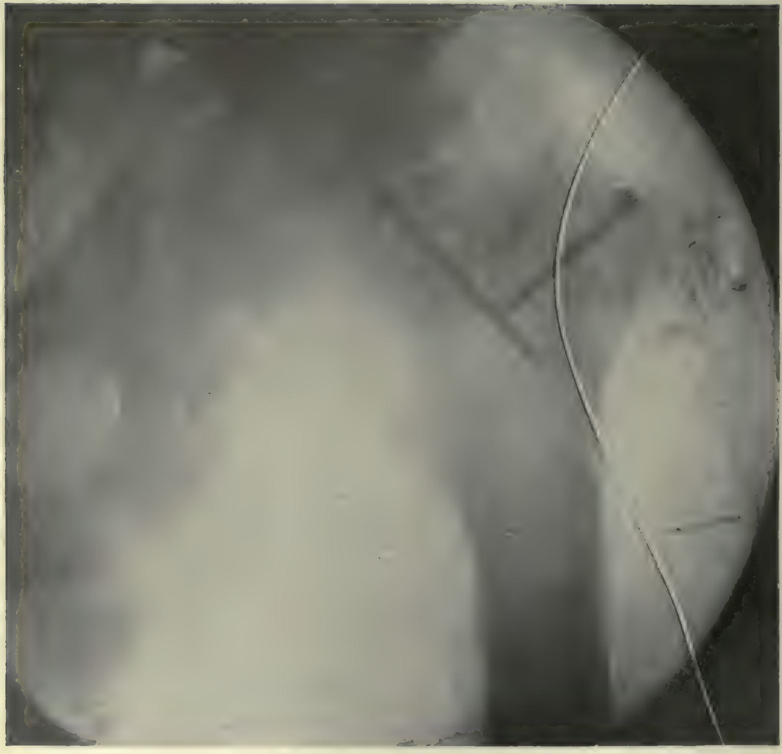


Fig. 4.—Old fracture of anatomic neck of femur. Absorption of head of bone and non-union. Four months after operation. A portion of the greater trochanter was removed and nailed to the femur to make a new head. The result was perfect. A firm bony union took place, with restoration of function in the hip. Full motion in joint, even to the extent of crossing his legs.

months afterward the neck has absorbed completely clear out to the shaft, leaving only a button of bone in the acetabulum on an exact level with the epiphyseal line. It will be noted in the first picture that the fracture occurred near the epiphyseal line and that the absorption of the neck has taken place from within outward, which is the characteristic course for this absorption.

If the fracture had occurred near the trochanter in place of near the epiphyseal line of the hip, no absorption at all would have taken place. Looking over the skiagrams in the pathologic museum we can see that that has been the course of absorption for all time. In this case the various later skiagrams show the progress of the absorption toward the trochanters. If this limb has been completely immobilized in an abducted position, either by nailing or otherwise, primary union would have taken place without shortening and without bone absorption. It seems to me that the friction of the neck on the fragment of the head plays an important rôle in the absorption process, as well as the direction of the circulatory supply as accentuated in a paper I read before the Western Surgical Association about three years ago. The man was forty-eight years of age.

Case 2 (Fig. 4).—Here is another case. This man had a fracture of the anatomic neck of his femur and of his pelvis. The skiagram showed that the head of the bone had become completely absorbed. In this case we removed about four-fifths of the trochanter and nailed it to the neck of the bone from within outward with one wire nail to aid in regenerating the head. That man is an engineer and is back at work, which means that we got perfect results. He has splendid use of his leg. He can abduct, adduct, and circumduct it. He can cross that leg over the other and put his full weight on it. The transplanted trochanter filled in with new bone, which took on exactly the same size and shape as a normal femoral head. That gives you an idea of how a new head can be made from the trochanter. In fractures of the neck of the femur it takes a much longer time to get union of the head and neck than in an ordinary fracture.

These patients must be immobilized for twelve weeks in order to get the best results. In children it does not take so long, but the majority of these cases occur in people between fifty and fifty-five.

Case 3 (Figs. 5–9).—This case was one of fracture well up in the neck of the femur, with strangulation of the ligamentum teres and necrosis of the head. It was dead, but aseptic. We nailed back in place and a new head was regenerated. You can see the result in the skiagram made four years after the operation, showing how the head has regenerated and filled in with new bone. The epiphysis was not destroyed. Bone growth is taking place from it. This patient was sixteen years of age. We operated on him seven weeks after the injury. He has full use of his hip-joint, with only about ¾ inch of shortening. In fact, he has greater mobility in this hip than in the other. Even when he is walking rapidly he does not limp. It is now four years since the operation.

These are typical results. We dislike very much to do these operations in older people, but we are supported by the work Mr. Lane has been doing and in which he got such good results.

[Mr. Lane is ready to begin the operation.]

MR. LANE: Dr. Murphy has asked me to explain to you how I get these bones into apposition. It is easily done. There is absolutely no difficulty, because you simply grasp the leg between these two powerful bone-holding forceps so that both ends of the bone stick right out of the leg. Some one turns the leg and the fragments come into apposition. If you train yourself to do it with these instruments, instead of trying to do it with brute force, it is a very simple operation.

In the case of a fracture of the femur with four inches of shortening elevate the limb, the fragments are shoved out, as I did in this instance, and they come into apposition. Then put in a plate. I am very glad to do this operation, but Dr. Murphy would do it a thousand times better than I could.

OPERATION

MR. LANE: On exposing the bone, it was found that there was a displacement laterally of about one-half inch. It was fairly and firmly fixed with a considerable callus. The callus was

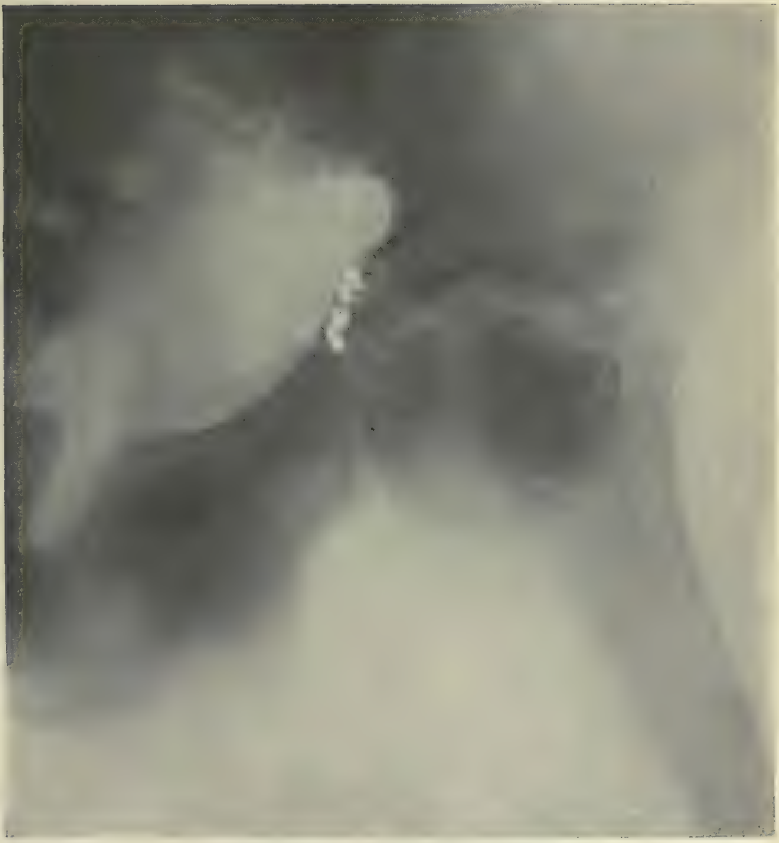


Fig. 5.—Fracture of neck of femur with separation of head, seven weeks after injury. Owing to separation of head from source of nourishment, it was dead but aseptic, so that it could be used to bring about the formation of a new head. The globular mass in the center of the picture is the head of the femur. The shaft extends behind and above it.

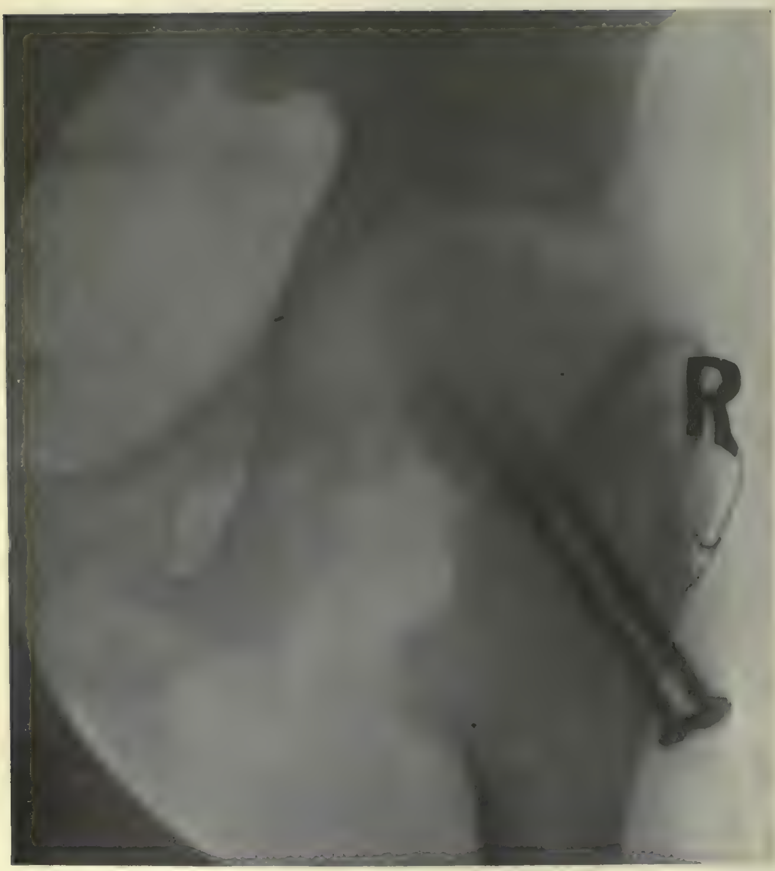


Fig. 6.—Head nailed to femur after reposition of fragments; beginning of regeneration of the new head.

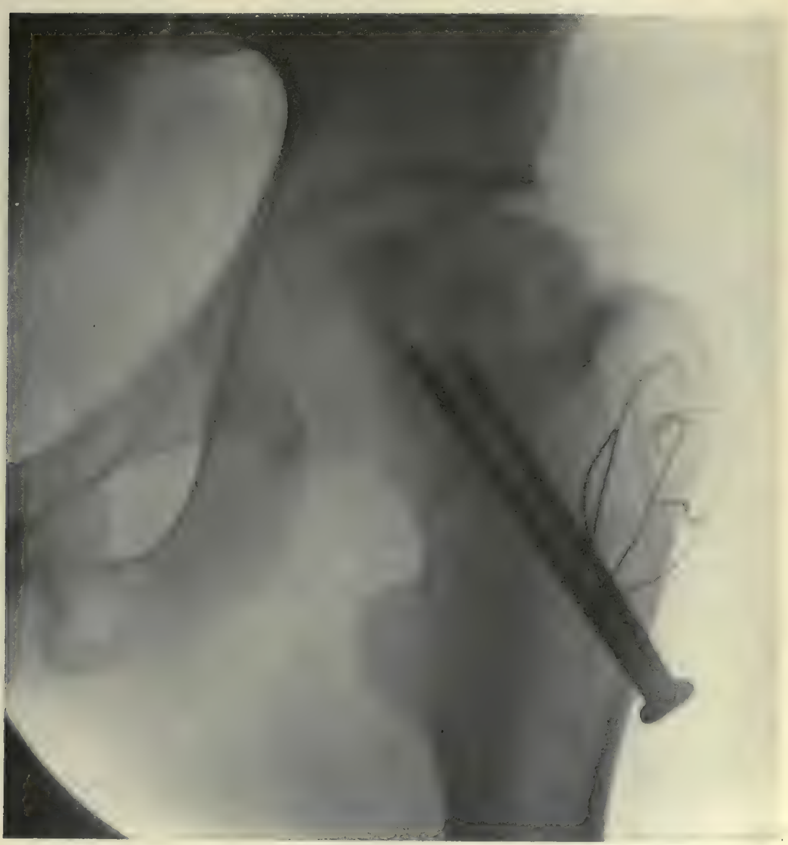


Fig. 7.—Skiagram of same patient taken two and one-half years after operation, showing nails still in place and also the two phosphorus-bronze wire loops which were used to suture the muscle aponeurosis. Bony union complete. By comparing this skiagram with the previous skiagram, taken shortly after operation, it will be seen there is epiphyseal activity, and that the head of the bone is enlarging to meet the demands made by the growth of the patient.

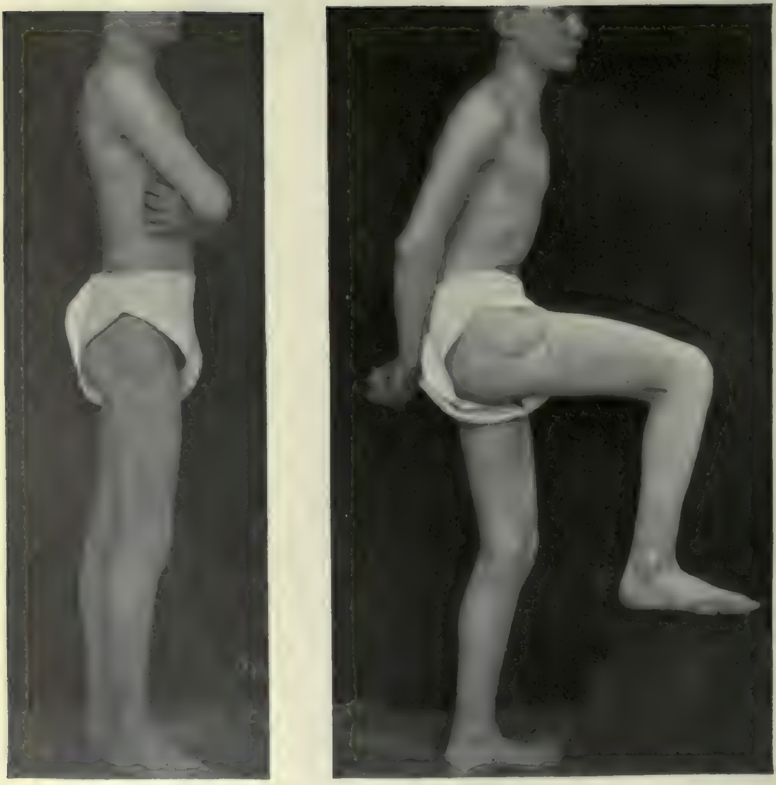


Fig. 8.—Three months after operation patient was able to stand erect with both feet firmly planted on the ground and little evidence of shortening. Flexion was possible at right angles or 90 degrees.

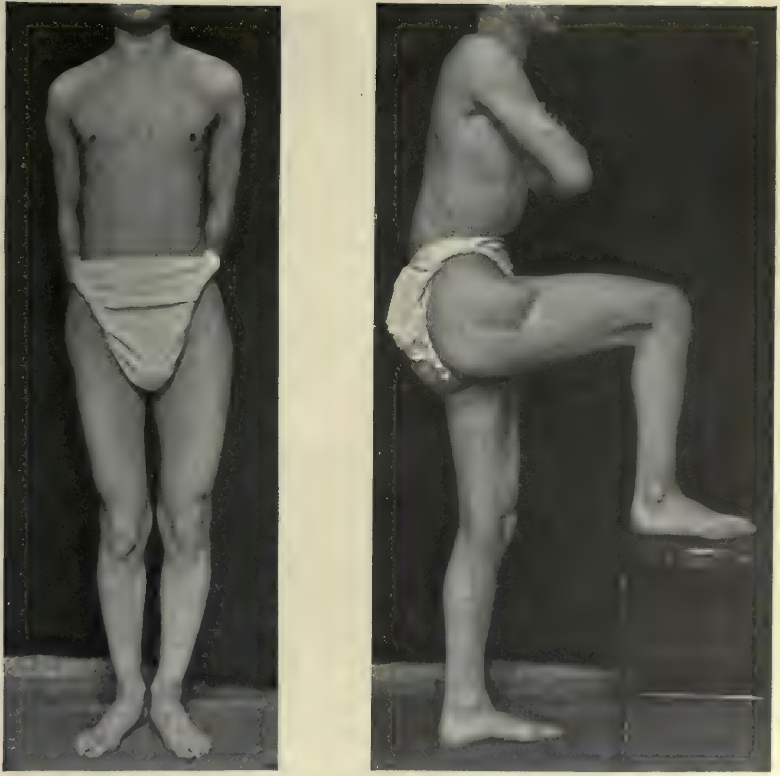


Fig. 9.—Two and one-half years after operation motion in hip-joint was normal—if anything, better than in the fellow-joint; even circumduction was full and easily done. There is a slight shortening of the leg of about three-fourths of an inch. When patient has shoe on he does not limp nor is shortening apparent.

first freed, and the line of fracture clearly demonstrated. Then it was depressed and then separated, and the ends of bone mobilized with a lion-jawed forceps. The ends of the bone were swung around into apposition and clamped there with our large clamp. The plate was slid under the clamp and attached and screwed to the lower fragment, and then screwed to the upper fragment, which brought about a most perfect apposition of the fractured ends of the bone. The screws and sponges used in the operation were never permitted to touch the hand or to touch any instrument which had touched the glove or the surface of the patient. The fact that no instrument that was touched by the hand was used insured the most perfect asepsis from the instrumentation and handling standpoint. This was accentuated as one of the most important factors in the operation, and unless exactions in detail in these particulars are attained and regularly attained, the operation will not be a success. I am not responsible for the defects of technic by others. No catgut ligatures were used and no catgut sutures. The skin was approximated with Michel clips.

Dr. Murphy: Do you put in muscle sutures?

Mr. Lane: No, nothing at all. The skin wound is closed with Michel skin clips.

Dr. Murphy: I am sure Mr. Lane will pardon my saying a few words on this operation. You can all see what has made his work in this line such a great success. In watching that operation, how many in this audience—and I know you are all able men—could handle all those instruments and never touch one of them with the gloved hand or allow it to come in contact with the edge of the wound? That is the secret of Mr. Lane's success. He is a past master of technic either in bone work or in abdominal surgery. He has learned how to use instruments, and never allow them to touch the wound. This case was a difficult reduction for two reasons, first, because of the position of the fragments, and, second, he did not have all the instruments he is accustomed to use at home. Notwithstanding these difficulties, he succeeded in putting those fragments into perfect apposition without touching the skin with an instrument or glove. I noticed him many times sending back instruments which the assistants, well trained under me, had handled. They simply erred in carrying out Mr. Lane's technic. That is one of the things that must be understood, and one of the things that must be carried out properly if you are going to get results in this work. This work can be done by any skilled surgeon, but every detail of the operation must be mastered, and to get results it must be done by men who have trained themselves to do it.

You note that he does not make an apposition of the muscles with sutures, but he is making an apposition of the skin with the Michel clips. The apposition of those fragments was perfect. The small, almost detached fragment was held by only a small piece of periosteum. It did not matter to him whether he left that in or not, so great is his confidence in his asepsis. If that bone remains sterile, it does not matter whether it is contacting with periosteum or whether it is living or dead, it will be regenerated as long as it is contacting with living bone. You can see the perfect alinement of that bone now that the plate is in place.

MR. LANE: Dr. Murphy makes me feel quite unhappy. I do not recognize my poor humble self in his beautiful description. It is nice to hear some one say beautiful things about you. I have been so much exposed to criticism that it is very nice that a man like Dr. Murphy, who can do bone work better than any man in the world, says such things about me.

DR. MURPHY (December 6, 1912): This is the patient that was operated on by Mr. Lane at the clinic November 23d, and today the dressings were removed and the Michel clips taken off. There was a complete and perfect primary union. The patient's temperature has never reached 100° F. at any time since the operation.

All of this, gentlemen, shows the effect of the superior aseptic technic carried out by Mr. Lane in the operation, even with strange assistants and in a strange operating-room. One had to see this operation in order to appreciate how all this work can be done by instrumentation and handless contacting of the tissues. We are all edified as well as educated by his work.

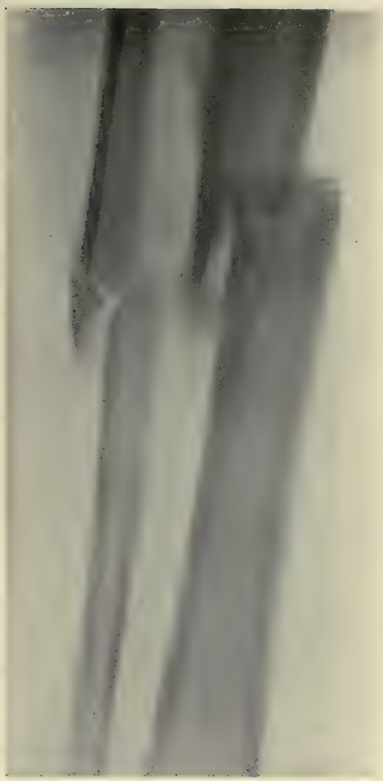


Fig. 10.—Fracture of tibia and fibula just below junction of middle and upper third, with upward and inward displacement of lower fragment of tibia. A small separate fragment can be seen between the main fragments of both bones (anteroposterior view).

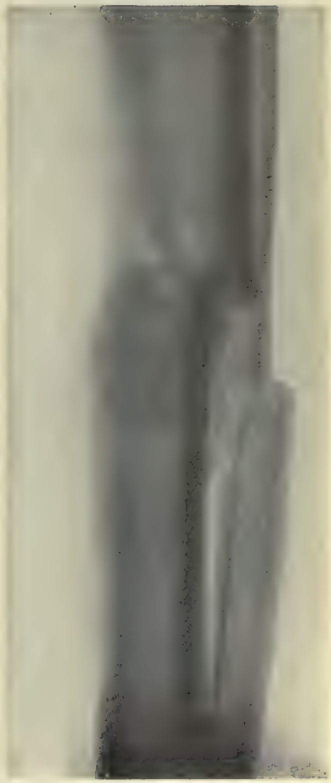


Fig. 11.—Lateral view of same case with little apparent deformity.

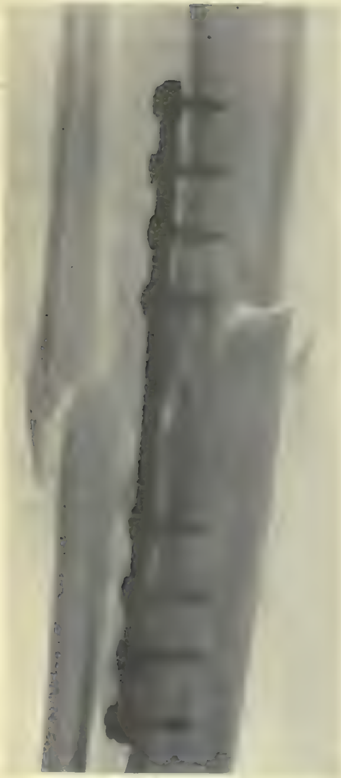


Fig. 12.—Anterior view after operation. The tibia only was splinted with an eight-screw Lane plate after both bones had been brought into alinement. The sequestra were not removed. The tibial sequestrum is firmly held against the main fragments by the bone plate.

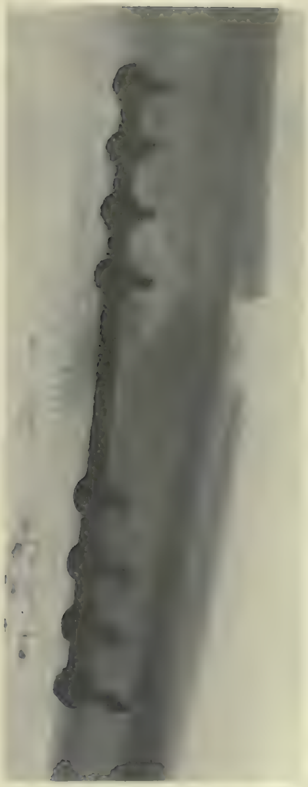


Fig. 13.—Lateral view of same case, showing perfect alinement of tibia.

OSTEITIS OF FEMUR

HISTORY

PATIENT, an unmarried female, aged thirty-two years, comes to hospital complaining of pains in left femur as a sequence to an acute osteomyelitis. Present trouble began when twelve years of age. On Sunday evening, September 10, 1892, she felt sick, had headache and general malaise. The next day she had several mild chills and that afternoon she complained of a severe acute pain over the lower end of the left femur, extending from just above, but not in, the knee-joint to the hip. That night she developed a high fever (?) and was delirious most of the time for two weeks. Does not remember how high her temperature was. Two days after the onset of the trouble her limb became swollen. The swelling increased in size, and opened of itself at the end of two weeks, discharging pus freely. As soon as the abscess opened the temperature dropped (?) and the pains became less severe. She does not remember having a sore throat, carious teeth, or any skin lesion at this time; no gastro-intestinal trouble and no vaginal discharge. In March following the onset an incision was made over the sinus above the external condyle, the wound was packed with gauze and allowed to heal by granulation. Was healed up in three months. A year later the sinus opened again and discharged pus. Bone was scraped, and wound allowed to granulate from the bottom. Remained healed for one and a half years, when it again broke open and a similar operation was performed. It then remained healed for nine years, but she had considerable pain in the winter season. At the end of this time it broke open again, and spicules of bone were discharged. After a short time the sinus closed again. Six months later she complained of a deep boring pain, worse at night, over the lower part of the femur. An operation was performed, but she does not know exactly what was done. Since then she has always had more or less pain, worse at times, usually in the winter season, and sometimes requiring morphin to give her relief. Her knee-joint has never given her any trouble and is now freely movable.

Examination shows an old, non-adherent scar over the lower third of femur on the outer side; tenderness on percussion over lower half of femur. Patient is in good health otherwise. Heart and lungs normal. Menstrual history negative.

COMMENTS AND OPERATION

Dr. Murphy: What date was that?

The Intern: September 10th, twenty years ago.

Dr. Murphy: Think of that! Where did the pus escape? Where was the sinus?

The Intern: On the outer side of the lower end of the femur.

Dr. Murphy: How far from the knee?

The Intern: Two inches above the knee-joint.

Dr. Murphy: It remained healed how long?

The Intern: Nine years. The blood count is negative. White cells, 9800.

Dr. Murphy: Is it negative with a white-cell count of 9800? Is that negative? Not at all. Positive, and positively wrong, too. If she has that count for three days,—and the count should be made for three days,—when it is that way it is a matter of great importance. She has an infection here that has been present for twenty years, has it not?

The Intern: Yes, doctor.

Dr. Murphy: What's the matter with her? What is she here for?

The Intern: To get relief from the pain in her femur.

Dr. Murphy: Where?

The Intern: The lower third.

Dr. Murphy: How often has she this pain?

The Intern: It is constantly present and is worse in cold weather.

Dr. Murphy: She has periodic attacks of pain, and it gets

Fig. 14.—Osteitis of femur, with great thickening and hardening of the bone. Numerous small foci of infected granulation tissue were scattered throughout the external lateral aspect of the shaft, from the junction of the upper with the middle third downward, but not involving the knee-joint.

so bad that she has to have morphin. She had the recurrence of pain, and the pain was so severe that she had to have morphin to get relief. That tells the story. Now let us look at the skiagram. It does not give us any particular information. There is no evidence of a distinct, clean-cut abscess, but she has an osteitis extending the entire length of her femur. What kind of osteitis is it? Is it a tuberculous osteitis?

The next question that arises is, What was the matter with her originally? What was her original lesion? Did she have a tuberculosis or a pus infection in the metaphysis of the femur? The history tells the story. She got sick on a definite day. She did not get sick in September, but on the tenth of September. A tuberculous patient never gets sick on a definite day—never! It is three weeks and a half or six weeks from the onset of their inconvenience before they are incapacitated. They are inconvenienced before they are incapacitated, while in osteomyelitis they are incapacitated inside of twenty-four or forty-eight hours, and usually inside of twenty-four hours.

In making the incision I will follow the line of the old incision, keeping to the posterior margin of the quadriceps extensor muscle—the vastus externus portion of it. I will not cut the muscle-fibers at all—I separate them. I will go along in that fascial fold all the way. There is the most common position of resistance to pus in the femur, and by going in laterally I am keeping anterior to the external popliteal nerve. It is right under my finger. There is one thing you must always remember when you are doing work in this neighborhood, that if you disturb that external popliteal nerve you disturb all the muscles of the leg which it supplies. There is nothing to be seen in the skiagram that one could interpret as a definite circumscribed abscess. There is a diffuse enlargement of the femur, with probably many foci of granulation scattered along the entire length of the bone. The irregularity in the bone and the thickened periosteum are very apparent. That shows the nature of the disease in this case. There [indicating] is a large exostosis, larger than the others, meaning what? That there has been a focus of infection in the vicinity, and that it emptied itself beneath the periosteum and started to produce an ossification in the muscle. Whenever you see this in the skiagram you know what has happened, and that it is not a periostitis, but an osteitis with a subperiosteal escape of infective products. That has been the greatest point of attack, the most common site of infection and of recurrence of infection. There was early destruction of the bone here. This particular spot is the triangular space between the heads of the gastrocnemius muscle. We will drill into the bone in several different places.

This is the hardest kind of work, because there is so much cicatricial tissue here. We have cicatricial bone, just as we have cicatricial tissue of the soft parts. The gray granulation tissue present here indicates that this is the chronic type of osteitis. That means that we will have to remove a great deal of bone. It is a severe type of osteitis from a pathologic standpoint, and a very discouraging one from a surgical standpoint—still more so from the patient's standpoint. We will remove all this granulation tissue and cicatricial tissue with the chisel. This bone is solid from periphery through to the center. It is not going to be soft until we get to the upper limit of the affected zone. The diseased spots in the bone are many, and they are the cause of her pain. Being surrounded by dense bone, the infective products cannot escape. That condition also causes this thickening of the bone and the change in its conformation. One sees the little pockets all through the bone and no medulla. This is a perfect picture of this type of lesion.

There is no central canal here. The bone is one solid mass. This disease burrows through the bone very much like a mole goes through the ground; it burrows out and burrows in, destroying the bone as it proceeds. I will drill down to the center of the bone. There is no disease focus there, and still when I follow that little red streak, which is the guide to the line of advancement, it leads to areas of bone destruction. There is a great deal of cicatricial bone here, but no infection at this time. No matter where I drill, the bone is hard, like an exostosis. This is exactly in consonance with the picture, showing that it is a subperipheral lesion of long standing, characterized by destruction and the formation of new hard bone. We will remove all this débris with the sharp spoon. I kept well within the cicatricial line, hence there is practically no bleeding at all.

We have cleaned out all of the disease foci along about 10 inches of the length of the bone on its external lateral aspect. We will now swab the wound thoroughly with a 5 per cent. aqueous phenol solution, so as to render the field of operation as sterile as possible.

The aponeurosis of the quadriceps muscle will be firmly reunited with plain catgut. We need not fear the occurrence of a hernia of that muscle. I take out this cicatricial tissue because if I leave it here I am likely to have failure of union. The skin incision will be closed with horsehair. Now read that history again.

[The intern reads.]

Dr. Murphy: Pus was discharged where?

The Intern: Outside.

Dr. Murphy: Outside of what?

The Intern: From the lower third of the femur.

Dr. Murphy: All right, put that down.

[The intern reads.]

Dr. Murphy: Why did you say, doctor, that it was an acute infective osteomyelitis?

The Intern: On account of the suddenness of its onset.

Dr. Murphy: What do you mean by that? What are the symptoms of an acute infective osteomyelitis?

The Intern: A sudden chill after exposure.

Dr. Murphy: That onset is just the same as that of acute pneumonia. All right, give me the syndrome of symptoms.

The Intern: Exposure to cold, sudden chill, followed by pain.

Dr. Murphy: Give symptoms in the order of their appearance.

The Intern: Sudden onset of pain in the femur, followed by a chill.

Dr. Murphy: Where does it usually localize?

The Intern: At the end of the long bones.

Dr. Murphy: What do you mean by "in the long bones"?

The Intern: At the epiphysis.

Dr. Murphy: No, sir. Never in the epiphysis. You never have an acute infective osteomyelitis occurring in the epiphysis on the shaft side. What is the end of the shaft called near the epiphyseal line?

The Intern: The metaphysis.

Dr. Murphy: That's right. The pain is usually in the metaphysis. If you will read the literature of acute infective osteomyelitis in children, you will find that the regular name formerly given it in the English literature is acute epiphyseal infection of the bone. They do not differentiate between an infection that occurs in the epiphysis and an infection that occurs in the metaphysis. The metaphysis is the place where this infection occurs with the greatest frequency. It may occur in the shaft of the long bone, but only very rarely. The infection may spread from the metaphysis to the shaft. Within a few hours after its appearance in the upper end of the tibia it may extend to the lower end of the tibia, extending from one epiphyseal line to the other, and producing an acute infection of the medullary zone.

Let us analyze the symptoms as they occur, and let us see the pathology present. Remember, symptoms are only expressions of pathologic changes, and the interpretation of symptoms into their definite pathologic entities is the *Ultima Thule* in the practice of medicine. That is the most important and greatest achievement in the practice of medicine; that is, the interpretation of symptoms and signs into definite pathologic entities.

The first element in the etiology shows what ? The exposure or trauma. The following night the patient has a chill and then wakes up in the morning with a high temperature and has aches and pains. She tries to get up out of bed, but cannot on account of the pain in the lower extremities. The fever runs up to 103° to 105° F. following that chill. What comes next? Pain—severe pressure pain; a severe boring pain. It is in the neighborhood of a joint, not in the joint, and it is of a boring character. The patient is intensely ill. Go on, doctor.

The Intern: There are swelling of the limb and edema.

Dr. Murphy: When?

The Intern: The swelling usually comes on in a few days.

Dr. Murphy: When?

The Intern: After the onset of the pain.

Dr. Murphy: Yes. It is not an early symptom at all. The swelling is never present as an early manifestation—absolutely not. Why? Because the infection is confined within that unyielding bony wall, and the periosteum and the tissue outside of that are not infected. There is never a primary swelling. I will show you wherein that differs from metastatic infection in the joint. Ninety-five per cent. of the cases of acute infective osteomyelitis are diagnosed as "rheumatism." If you are going to do anything at all for that patient in a curative or preventive way, you should do it then, do it early, but, as a rule, during the early and important hours the patient is being treated for "rheumatism." Why? Because the pain occurs near a joint. Then you treat it as a metastatic synovitis because the trouble began with a chill, and we have learned that a synovitis associated with a chill is of a wicked type. What else?

The Intern: The leukocyte count.

Dr. Murphy: Yes. The leukocyte count is high. It usually runs up to 20,000, 30,000, and 40,000 because the medullary tissue in the bone is infected; the cancellous tissue is infected, and the products of infection are held in it under the highest possible tension. The pain is severe and continuous. The intoxication is great; the toxins get into the circulation, and the patient has a temperature of even 105° to 106° F. How long does this state of affairs continue—the high temperature and the pain?

The Intern: While the pus is under tension—until such time as it comes out of the bone.

Dr. Murphy: Yes. All right. Until such time as the pus comes out of the bone and spreads along the outer surface of the bone beneath the periosteum, and then for the first time there is local sensitiveness to superficial pressure. Go on, doctor. What next?

THE INTERN: Then the swelling.

DR. MURPHY: Yes. As soon as the pus ruptures through the periosteum into the cellular tissue, then the swelling appears; a phlegmon has formed and it will perforate through the skin sooner or later, and then the patient gets relief with the subsidence of his symptoms—the pain, the tenderness, and the high fever. Now, doctor, what about the neighboring joint?

THE INTERN: After exposure or trauma the patient has a sudden chill, followed by fever, with pain and localized pain.

DR. MURPHY: Where?

THE INTERN: In the joint infected.

DR. MURPHY: Go on, doctor.

THE INTERN: In metastatic arthritis there is limitation of motion—usually an ankylosis.

DR. MURPHY: We are not within six or eight weeks of that time. You cannot jump that span. What is happening all this time. We have considered only the initial symptoms of osteomyelitis up to the time when the pus burrows through the bone and appears beneath the periosteum. That gave us the external signs of the lesion and the tenderness. If the patient is young and the disease is in the tibia, after the pus gets under the periosteum it may travel from the knee to the ankle. It just lifts the periosteum up from the bone all the way; or it may travel from the knee to the ankle in the medulla. When it does, what happens? The circulation is cut off from that bone by the destruction of the medulla and elevation of the periosteum, and finally there occurs a necrosis of the entire shaft of the bone. If not complete, there is as much bone destroyed as is penetrated by the pus until the periosteum is lifted up from the bone and the pus makes its escape from beneath the periosteum into the cellular tissue. Sometimes it is just a portion of the metaphysis that is involved; sometimes it is a little round abscess; or it may involve the entire shaft; more commonly the entire shaft is involved in children rather than in adults, but still the entire shaft may be destroyed in adults as a result of an acute infective osteomyelitis. Why does the disease destroy the whole bone?

The Intern: Because the blood-supply is cut off.

Dr. Murphy: Why does it travel the full length of the bone within the medulla? The medulla is the least resistant part of the bone during the early stage. The infection extends from the medulla, and when it comes beneath the periosteum, it lifts up the periosteum. It makes space for itself in the medulla and under the periosteum. Why do I want to make that particularly impressive? Because that is the key to the treatment. If you know why you are doing things, it is no trouble to do them. How long a time is required for the entire shaft to be destroyed?

The Intern: Depending on the acuteness of the infection.

Dr. Murphy: What is the rule?

The Intern: Three or four days after the onset of the disease.

Dr. Murphy: Not so indefinite. The rule is for the maximum of destruction to be produced inside of seventy-two hours. The rule holds just as good here as it is to have the maximum degree of gangrene of the appendix produced inside of forty hours. Gangrene of the appendix rarely occurs after forty hours. If it does not occur then, the appendix is going to survive the infection without becoming gangrenous. In the acute infections of the bone the maximum of destruction generally is produced within seventy-two hours, and the major portion of that destruction has taken place in half that time—in thirty-six hours. What does that mean to us? It means that if we are going to be of the greatest benefit to the patient, we must do something before the end of thirty-six hours. That's what it means. It is the appendiceal proposition all over again. In connection with appendicitis we have learned that any case of appendicitis that goes on to the development of pus outside of the appendix has been mistreated. The gangrene is incontrovertible evidence of that, and no explanations avail. We have learned the lesson about appendicitis, but we have not learned the lesson so well about osteomyelitis. If we are going to benefit the patient, we must diagnose that osteomyelitis and do the things that are necessary to be done before the lapse of thirty-six hours, or, better, before the lapse of twenty-four hours, and, still better, before the lapse of twelve hours.

Now let us go over the symptomatology and pathology of this infection. What has the pain to do with it? What has the chill to do with it? What is happening when the patient has a chill?

THE INTERN: The chill shows that there has been an infection in some other place in the body which has been metastasized in the bone.

DR. MURPHY: That is, when the microörganisms are circulating in the blood and are deposited in the medullary tissue of the bone and begin to multiply it causes a chill. The chill is the expression of the absorption of the products of infection, and it occurs very early. The elevation of temperature is the expression of the absorption of the products of infection and occurs very early in osteomyelitis because the pus is under such great tension. If that infection has been arrested in the metaphysis of the bone, what does it do after it is so arrested? It infiltrates the medullary tissue first, then gets into the Haversian canals and canaliculi, and then penetrates the bone lamellæ and that penetration and the toxic action of the microörganism produce a necrosis and death of the bone, because the infective products are held under tension.

What does the dentist do when a patient comes to him with a toothache? The moment the dentist touches that tooth he knows, no matter whether the patient has an elevation of temperature or not, that there is an infection at the root of that tooth. He drills into the tooth with a bur and relieves the tension caused by the confined infective products. As soon as the tension is relieved by a rupture of the periosteum and the exit of the infective products on the surface of the limb the absorption of toxins ceases and the pain subsides. That is what we must do if we wish to treat an acute infective osteomyelitis to the best possible advantage. We must release that infective focus in the bone and do it before the lapse of thirty-six hours.

I will cite an instance that occurred at a meeting of the American Surgical Association, which is the master surgical association in this country. A doctor came to that meeting and brought with him seven shafts of long bones, trophies of what? Trophies of the most incompetent management of acute infective osteomyelitis. Why? Because if these infections had been vented early; if that infective focus in the bone had been opened early, there would be no shafts to display to the association; there would not have been any destruction of the shaft which crippled and permanently incapacitated the patients. All we need to do in these cases is to do exactly what the dentist does, cut down on the bone, and you need only a pocket instrument-case to do this, and bore a hole into the bone to relieve the patient of his symptoms and to save his bone from necrosis.

This case was not of the regular type. The infection stayed near the surface all the time and produced a gradual sclerosing osteitis. It was not a malignant type of infection. Whenever this infection reaches the periosteum she has the greatest pain. When it ruptures through the periosteum, she gets sensitiveness to pressure and edema and swelling. That clinical picture is classic of this disease, whether it is of the burrowing type or of the circumscribed abscess type.

The lesson we must learn from the clinical history of every case of acute infective osteomyelitis is that it begins with a chill; they have pain, indefinite in location, usually near a joint, never associated with an acute joint effusion, and therein you differentiate it from the arthritides called "rheumatism," or a metastatic arthritis, if you will. If you are going to be of real service to the patient, you must open that pus cavity inside of thirty-six hours and probably inside of twenty-four hours. You can make a differential diagnosis of the location of the pathologic process if you will carefully observe these symptoms, clinical course and physical signs, and you can avoid destruction of the bone, with the subsequent deformities of the limb and the ankylosis of the joint which occur so often following infection of this kind.

We will dress this wound with moist 5 per cent. phenol gauze. The next dressing will not be made before two weeks from now, when the stitches will be removed. Subsequent dressings will be made at intervals of a week, or oftener if there is much discharge coming from the wound. We do not provide for drainage and we do not put on a plaster-of-Paris cast.

[This patient left the hospital nine weeks after the operation with the wound completely healed and free from all symptoms.—Ed.]

LUXATION OF SEMILUNAR CARTILAGE

HISTORY

Patient, a female, aged thirty-five years, comes to hospital on account of trouble in right knee-joint. June 6, 1911, while traveling on a train, her right limb "fell asleep." When she stood up, her knee "twisted," and she was immediately seized with an acute pain on the inner side of the knee-joint. Joint was locked, and only after considerable manipulating and rubbing could she move it again. She says she felt something "slip out of the joint as the pain came on." Two hours later, when she got off the train and was walking home, the same thing happened. Her knee was slightly swollen and painful for about two weeks. She did not use the limb during this time. July 11, 1911, as she was opening a trunk she heard something in her right knee-joint snap. Her knee became flexed, and she could not move it. She had to remain in bed three weeks. She had the knee massaged and hot dressings applied. After a time she could use it again. Since then she has had repeated attacks of locking and pain, but did not again feel anything slipping out of the joint. This knee is not so strong as the other, and she has pain in it almost continuously. There is tenderness on pressure over the internal semilunar cartilage, none over the external, with marked crepitation on moving the joint. Patient walks with a limp. Patella is not abnormal. Patient is otherwise healthy.

COMMENTS AND OPERATION

Dr. Murphy: She had repeated attacks of what?

The Intern: Of pain in and locking of the joint, but did not feel it snap.

Dr. Murphy: She could not hear it and feel it in the first attack. The doctor has that a little mixed. When the trouble occurred on the train, she could not get her limb into a straight position until she endeavored to walk home, and then she heard a sound similar to the one she heard when it went out; after that she could walk.

She always had to walk with a cane. If she did not, the trouble kept recurring, and, as a result, she now has a chronic synovitis. The semilunar cartilages are more commonly fractured than luxated. They usually fracture on the edge and split into a fine web. You will recall an article written by Rutherford Morison, of Newcastle, who has a larger experience in this line of work than any other man in the world. He has 500 specimens of semilunar cartilages which he has removed. He practises in the coal districts of England, and it is very common for coal-miners to have luxation of the internal semilunar cartilage. When that happens they are at once taken to the hospital, and within a few days the semilunar cartilage is removed. In this country that accident occurs in an entirely different class of people. It occurs in athletes and those who play golf and baseball. It is either becoming more common, or we are paying more attention to it. There is scarcely a time in which we have not one or more of these cases in the hospital, showing how common it is when recognized. If it is not recognized and treated properly, there are baneful results to the joint.

When first the patient has this lesion he has pain and lameness, and finally gets a chronic synovitis. You can hear a crackling noise when the patient walks across the floor. Then comes the tendency to metastatic infection, called "rheumatoid arthritis," and many cases of "rheumatoid arthritis" of the knee are simply old cases of luxation of the semilunar cartilage which have produced a fibrous synovitis, and the patient is a semi-invalid all the time.

These evil results emphasize the necessity of doing something to relieve these patients. First immobilize the limb and put it in a plaster-of-Paris or a leather cast, and keep the leg in that cast for a number of months. Some of the patients recover; at least, they go many years without having a recurrence. But in a large percentage of them, when once it occurs, whenever they put the limb in a certain position or in the same position as when the original luxation occurred, they have it again and again, and the palliative treatment is often kept up for two or three, or even four and five years. It is the uniform practice now to advise the removal of the cartilage. That involves a certain element of hazard, and the most serious hazard is the occurrence of infection in the joint. Fortunately, the hazard of infection is not so great as it would be if we opened a perfectly normal joint, because you have here a joint in a condition of chronic inflammation, and we know that in such a joint there is much less tendency to infection than where the conditions are normal and the tissues are not infiltrated, and the lymph-spaces coffer-dammed. In the early cases, unless there is considerable inflammatory reaction following the trauma to the cartilage, we aspirate and inject the joint with a 2 per cent. formalin in glycerin solution a week before the operation. In older cases we do not do that at all. Where we have a fracture of the patella the result of a severe trauma we do nothing at all. We just wait for a week or ten days until we know that the lymph-spaces in the tissues have been coffer-dammed; then we wire the patella. If, however, a patient fractures his patella as the result of his foot slipping and putting the patellar tendon on the stretch, we inject 10 or 20 c.c. of 2 per cent. solution of formalin in glycerin that has been mixed twenty-four hours, into the joint for the purpose of producing a reaction in the tissues which will coffer-dam the lymph-spaces. At the end of ten days all effusion will have subsided; then we open the joint and wire the patella. That very materially lessens the hazard of infection. Every one knows how much more difficult it is to infect a granulating wound than a fresh wound, and that is the basis on which these intra-articular injections are given.

One of the lesions that one can confound this with, without a careful examination, is an external luxation of the patella. These external luxations of the patella are not infrequent, and unless one studies the history of the case carefully and examines the patient thoroughly, a mistake in the diagnosis between these two conditions may be made. Perhaps you will recall a case we had in this clinic last year. The patient gave a history very similar to this one. She had had a number of attacks. On questioning her, she said that her joint locked, and that after a certain movement it snapped, and she went on about her work. In examining the case, as I always do, I found it was not an internal semilunar cartilage luxation at all—it was a luxation of the patella.

In making the incision in these cases we begin about three-quarters of an inch inside of the inner margin of the patella, in a line with the patellar ligament, extending the incision down to the fibrous capsule of the joint. Remember, this capsule does not stretch—it is inelastic—it tears, but it does not stretch. The care with which one must do all his work in bone and joint surgery is in marked contrast to what can be done in the surgery of the peritoneum. All manipulation and touching of tissues with hands, sponges, or instruments must be avoided as much as possible. I think the one man who accentuates that more than any other in his work, if not in words, is Mr. Lane, of London. We never allow anything to touch these tissues that is not known to be absolutely sterile. Sponges are used only once. They are passed in and out at once. They are not allowed to come in contact with the skin or anything else during their passage to the wound. They are handled only with sterile forceps, never with the gloved hand. The instruments are handled in the same way. They are touched only where they are held to be of use. The service portion of the instrument is not allowed to come in contact with anything but the wound. Such technic as this obviates most, if not all, of the danger of infection.

I want to show you the two layers of this capsule. I am not through the synovial membrane at all. I have only cut the fibrous layer. See how nicely that can be picked up. Now I cut through the synovial membrane. The semilunar cartilage slips in and out of the joint without the slightest trouble. I can even lift it clear outside, showing how perfectly free it has been in the joint. That is a clean-cut case, one which shows the pathology of this condition much better than does the average case. There was no attachment at all of the semilunar cartilage on the central side. That is the most mobile cartilage I have ever had. I succeeded in bringing out the whole circle of cartilage. That is not easy to do. The chronic inflammatory condition present here accounts for the grating mentioned by the patient.

The next thing to do is to suture the capsule. You will note that nothing touched that joint but my scissors and forceps. That is the key to success in the surgery of bones and joints. Now you can see that casts would not affect that condition at all, and that radical treatment was the only treatment that promised this girl any relief from her condition.

I will close the fibrous capsule very carefully with chromicized catgut sutures, so as to bring the synovial surfaces in contact. I have been careful not to abrade the synovial membrane, because if any of the lining endothelial cells are rubbed off, adhesions will form, and even a fibrous ankylosis may result.

Let the records show that the inner half of the semilunar cartilage was entirely detached, and that it was the outer half that was being traumatized. The outer half was slightly fixed. When she bent her knee in the superlative degree, the luxation occurred. She felt it snap and that threw it out. The skin incision is closed with horsehair and dusted with bismuth subiodid, a desiccating powder. It is not antiseptic.

VISITING DOCTOR: How is that leg dressed?

DR. MURPHY: It is dressed in a straight position, and placed in a wire cage—nothing else. We seal the wound with collodion gauze and put over that a 5 per cent. moist phenol gauze dressing. We have gone back to that dressing in all our joint and bone work, particularly bone work.

This patient will have a Buck's extension put on with 15 pounds weight attached. We do that in the case of every knee we open in order to prevent intra-articular pressure.

[The stitches were removed on the fourteenth day. Primary wound healing. The patient was discharged from the hospital after four weeks, with the knee in perfect condition, no pain, no tenderness, no swelling, and normal function.—ED.]

FLOATING CARTILAGE

HISTORY

Patient, a male, aged twenty-two years, comes to hospital on account of a loose body in the right knee-joint.

About the first week of January, 1912, he gave his knee a twist. He experienced a sudden sharp pain on the inside of the right knee, and a "buzzing" sensation in the joint. His knee did not become locked, and he was able to walk home. Two days later he noticed a swelling on the inner side of the knee. On palpating it he found that something slipped from beneath his fingers. The swelling soon subsided, but when he tried to walk, his knee became locked at every step and this body kept slipping in and out of the joint. Since then he has worn a tight bandage around the knee. This keeps the body in place, except when he is climbing stairs. The patient can find this body in the joint and push it out beneath the skin. Patient is in good health otherwise. He does not remember injuring this joint or having inflammatory trouble of any kind before the present trouble started.

COMMENTS AND OPERATION

Dr. Murphy: This is a floating cartilage and not a fractured cartilage. There are essentially three varieties of floating joint cartilages. First, there is the accretion cartilage, which begins as a separation of a fold of the serous membrane or a small fragment. That is the type we have here today. Second, a fragment of cartilage of considerable size is torn loose in the joint and floats about, but does not increase in size. Third, an attached portion, either of the synovial membrane or of the fibrous tissue, becomes traumatized in the joint and increases in size, but remains attached. It swings around in the joint and acts as a floating body. These are the three types of floating cartilage. They are all different from the type of luxated semilunar cartilage or fractured semilunar cartilage because a fractured semilunar cartilage, which is enormously more common than the luxated cartilage, is always attached at the central or peripheral zone.

You will remember the case we had a week ago today. A young lady was sitting down on her heel, when suddenly she was seized with pain in her knee-joint and it became swollen. You will remember in that case the internal semilunar cartilage was entirely detached on the central side and I could swing it in and out of the joint. A dislocated internal semilunar cartilage is enormously more common than we imagine. It causes a great deal of destruction to the joint. It causes a hyperplasia of the capsule, and the condition resembles a rheumatoid arthritis. The joint is incapacitated by the traumatic inflammation that occurs within it. The lesson we learn is this: When the cartilage once commences to be luxated, if the patient is fairly active there is but one line of treatment that is effective and safe, and that is the removal of the internal semilunar cartilage. The internal semilunar cartilage gets displaced fifty times to one displacement of the external cartilage. This statement is based on the experience of Rutherford Morison, of Leeds, England. He has had the largest experience in that line of any man in the world. He lives in the coal district, where the miners work in a stooping position, in low shafts, and that throws that cartilage out very frequently. The moment a workingman in the coal-mines in Newcastle has that type of injury, he is taken to the hospital, the joint is opened, and the internal semilunar cartilage is removed. Inside of three weeks he is at work again. The first able paper on this subject was written by Edmund Owen, of London. His patients were athletes. He put on casts and the patient would be relieved, but the condition recurred again and again, differing entirely from Rutherford Morison's experience, who thinks nothing of opening a joint, because he is so familiar with the work and is so dextrous that he does not infect the joint.

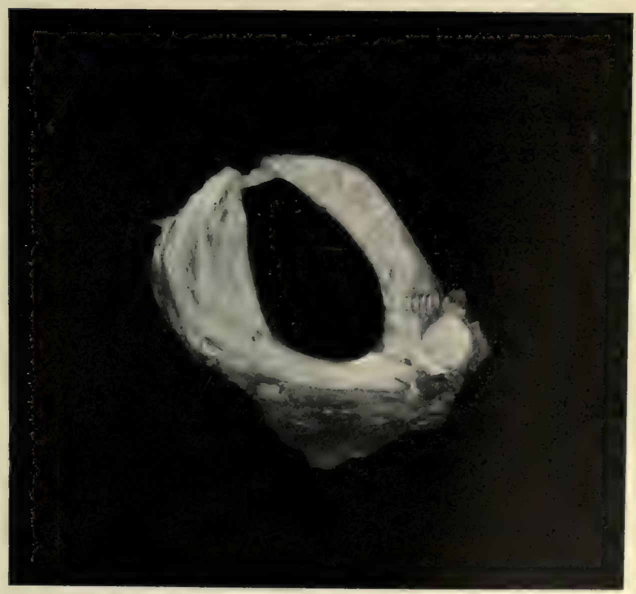


Fig. 15.—Internal semilunar cartilage removed in its entirety, showing thickening of one edge.

I have had the cartilage in this case out half a dozen times. Never cut down on that until you are able to locate it definitely, because you will otherwise traumatize the joint. In the management of these cases simply open the joint, remove the cartilage, and close the capsule with chromicized catgut. Only a small incision need be made directly over the foreign body. Grasp it firmly through the skin with finger and thumb, and then make the incision directly over it.

The application of ambulatory apparatus is deceptive to the doctor and to the patient; they have no real value, and it is not the treatment for this lesion. The right treatment is the removal of the cartilage, because if allowed to remain it keeps up a continual inflammation in the joint. This is the typical accretion variety of floating cartilage.

In suturing that capsule I include the synovial membrane in the suture. We will have no traumatic inflammation in this case. We did not touch a thing in that joint. We split the capsule, brought out the cartilage, and closed the incision in the capsule. We respect that joint. Neither sponge nor instrument touches the synovial membrane. A few horsehair sutures close the skin incision. Bismuth subiodid powder is dusted on it, and collodion gauze seals it. A 5 per cent. moist phenol gauze dressing is then put on. No cast and no extension.

I will bisect what we have removed and see what its construction is on the inside. The accretion type often is ossific in its center. This specimen is cartilage through and through. It is of typical accretion variety.

[The stitches were removed on the tenth day. Primary healing. Patient discharged from hospital in two weeks.—Ed.]

FECAL FISTULA FOLLOWING APPENDECTOMY

HISTORY

Patient, a little girl ten years of age, comes to hospital on account of a fecal fistula in appendicular region.

July 13, 1912, the patient was operated on for acute appendicitis of three days' standing. The appendix was found perforated. The wound was closed. Three weeks later there was a large collection of pus in the peritoneal cavity. A large drainage-tube was put in through an incision in the left iliac fossa, and brought out through an incision on the right side. A quantity of pus discharged. After four weeks the tube was removed, the left side healed up, but a fecal fistula formed on the right side. Since then the contents of the bowel have come through this opening. At present there is an opening in the abdominal wall the size of a silver dollar, where the drainage-tube had been. Through this opening the mucous membrane of the large bowel herniates. The hernia disappears within the abdomen when the patient lies down and retracts the abdominal wall. There is a high-grade dermatitis all around the opening. The mucous membrane that herniates through this opening is red and granular on the surface. Patient is in good health otherwise.

COMMENTS AND OPERATION

Dr. Murphy: When was the pus discovered in the peritoneal cavity?

The Intern: Three weeks after the wound was closed.

Dr. Murphy: On what day of the attack of appendicitis was she operated on?

The Intern: On the third day.

Dr. Murphy: Are you sure about that? Was it the third or fourth or fifth day?

The Intern: It was the third day, she says.

Dr. Murphy: What you want to ascertain is, was she operated on the first or second day. At that time there should be no pus outside of the appendix, and the closure of the wound was the proper thing. I received a reprint a few days ago in which the author speaks of the desirability of closing the peritoneal cavity immediately, even when there is pus present. He does not, however, make a differentiation between the various kinds of pus—between the protective or defensive pus and the offensive pus. We all know that long before there is a perforation of the appendix, early in the attack there is a large quantity of milky material in the peritoneal cavity which we call pus, but which we know is absolutely innocuous. It is a defensive material, filled with polymorphonuclear leukocytes, which give it the milky appearance. It is there for protective purposes. If the same quantity of pus is present on the third day after a perforation of the appendix, we would have an entirely different proposition to deal with. The doctor who wrote the article in question did not make that differentiation, and one should make that differentiation because we have exactly the same conditions to deal with here as in a perforation of the alimentary tract, or the pus of an old pus-tube, which, we know and have known for many years, is sterile, and which permitted Mr. Tait to make such splendid demonstrations to the English medical profession in the way of going in and taking out pus-tubes and letting large quantities of pus get all over everything.

This patient was not operated on in the pre-suppurative stage; in other words, it was not a timely operation—and what is a timely operation? It is the operation that gets that appendix out in the acute stage before the infection has passed beyond its wall. Every case of appendicitis that is allowed to go on to the stage of perforation is irrefutable evidence of bad medical management or that the patient or the patient's family delayed too long in accepting the doctor's advice to operate. All this has nothing to do with this case, except to bring out the fact that

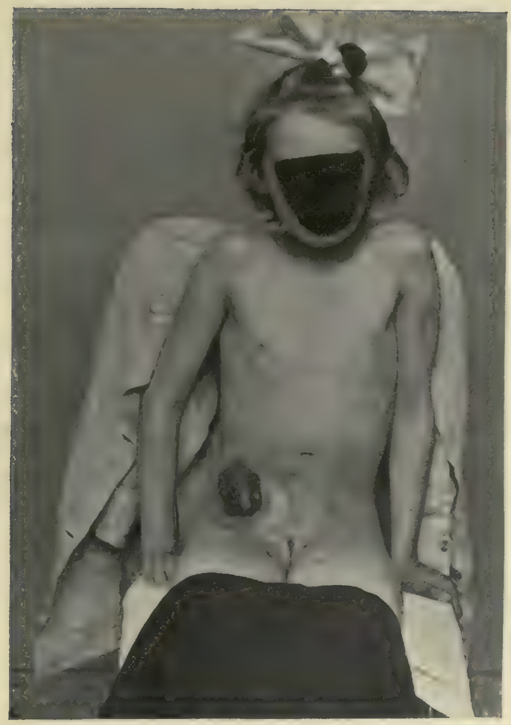


Fig. 16.—Hernia of the cecum. Extruded. Lumen of bowel barely visible in center of mass.

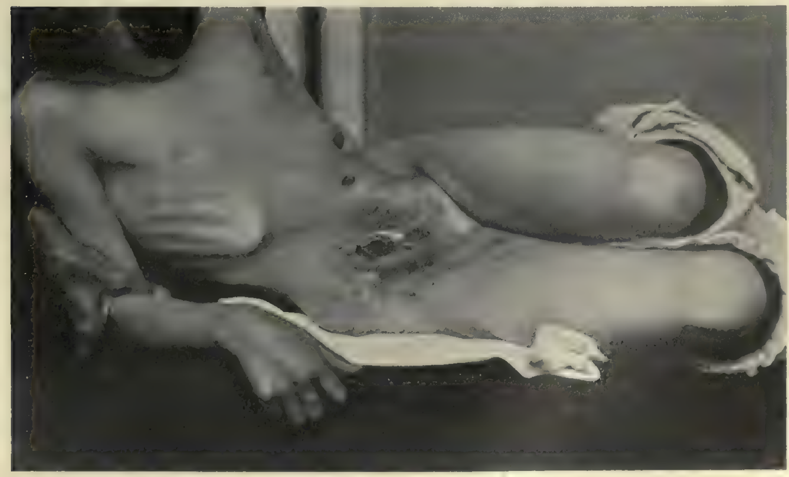


Fig. 17.—Hernia of the cecum reduced voluntarily by forced retraction of the abdominal wall. Severe degree of dermatitis all around the fistulous opening.

the operation was not done as early as it should have been done. When the patient was operated on there was some pus present, but it was believed that the pus was not virulent and the wound was closed.

There is now an ectropion of the mucosa of the cecum, not the intestine running in and out, and the part that has lost its gloss is the mucosa, not the peritoneal covering. It is firmly adherent around the margin of the opening in the abdominal wall, and the ileum presses forward under the cecum and everts its mucosa through a comparatively small opening. These cases are very difficult cases to handle—difficult, first, because we have the abdominal wall to close, and, second, there is a large opening in the large intestine which, on its posterior half, at least, has no serous covering, which means that we will not primarily have agglutination of the serous surfaces when we make the suture. It is further difficult to manage because we have an infected field to work in. For this reason not infrequently there is failure of union at the line of suture.

It is my belief that we can free the intestine along the line of adhesion and get a good place to put in our sutures. We will have to put in at least three rows of sutures, the first two rows being of Pagenstecher linen, and the outer one of plain catgut, because in all probability the wound will be infected. We will not close the wound without drainage because of the dermatitis which has been present for such a long period of time.

The management of an intestinal fistula is not an easy matter at all. It is a very difficult problem. An operation for intestinal fistula is one of the most serious operations, and in proceeding to attack an intestinal fistula you must have a well-outlined definite plan of procedure in mind. The first proposition is that you must expose the opening into the intestine. The second proposition is that you must free the intestinal wall from all the attachments in the neighborhood. It must be perfectly free. You cannot make a line of suture and leave the intestine attached to within half an inch of the line of suture. If you do, it will be a failure every single time. The intestine must be free and completely free from the abdominal wall, so that you can lift it out into the field if you so desire. Then, after it is out, insert your lines of suture; and where you have the large intestine to deal with and no peritoneum, it is very desirable to sew an omentum flap over the line of suture.

That is an unpleasant field to work in, and that is why we fear failure of union. This child is very expert in the handling of this hernia. As she strains she can force the bowel away out, and when she draws it in, you can look into the cecum. We can see the ileocecal valve. It is not pressed out far enough this morning. Yesterday she had it out so that one could see the ileocecal valve.

We will not disinfect the field of operation with a 3.5 per cent. tincture of iodin, as is our usual custom in this clinic. We use only sterile water and alcohol, because of the dermatitis present here. We have found that the full strength—7 per cent.—tincture of iodin occasionally causes a dermatitis, especially in patients susceptible to the irritant action of iodin. Therefore we use a 50 per cent. dilution or a 3.5 per cent. tincture.

The dry skin is painted with that just before the anesthetic is begun. Then it is covered with a sterile towel. The field is then ready for the operation.

I am going to endeavor, first, to detach this bowel from the abdominal wall, just at the margin of the mucosa. There is the free peritoneal cavity now underneath, not open, but you can see the peritoneum rise and fall. We must be exceedingly careful in freeing this bowel from its adhesions. We will first free the mucosa, so as not to open the peritoneal cavity. Now I have this mucosa ready for closure, using a straight needle threaded with Pagenstecher linen. I have no trouble in making an entropion of that cut edge of the mucosa. Now I have all that raw surface of mucosa turned into the bowel and the bowel is closed.

The next step is to open the peritoneal cavity and detach the remainder of the intestine from its fixation. This is done with less difficulty than we experienced in freeing the mucosa. Now we commence to pull up the intestine. There is the free peritoneal cavity. Now we have the lower side fairly well freed.

We come along now on the outer side, where we expect to have no peritoneum, and free that adhesion. That is what I mean by freeing the intestine from its fixation to the margin of the fistula. Now the intestine is perfectly free. There is the part of the intestine that has no peritoneum on it. It shows there very nicely. Now we will put in two rows of so-called Lembert stitches, but we have no serosa.

Visiting Doctor: Was that first stitch linen?

Dr. Murphy: The first row of stitches was linen and the second is also linen. The third will be a catgut stitch.

This is the peritoneum you see quite a little distance back. This fatty tissue in here, which gets its vascularization from above, we will not interfere with at all further than to free it and permit me to get a better line of overlapping. The weakest point in every gastro-intestinal anastomosis is the angle. If you go over the literature of gastro-intestinal approximation, you will find that Billroth and Wölfler had their trouble with the mesenteric angle. The temptation is very great to close that wound without a drainage-tube, but you remember we learned when we were young to "yield not to temptation." We therefore put in a rubber drainage-tube external to the cecum and passing downward. I am getting my muscular flap ready, and I am going to make it of the overlapping type. All that work is practically done extraperitoneally.

There is the aponeurosis now of the external oblique muscle in the field. I am making my imbrication of the muscle-flap now, using catgut for my suture material.

The greatest danger of infection is going to be right in this cellular tissue. Cellular tissue does not resist contact with colon bacilli. It resists other microörganisms very well, but not colon bacilli. All the knots are placed on the outside. Lysol and phenol are the best germicides for the colon bacillus.

Let the record show that the sponges have been counted and found correct. We had no sponges in the abdominal cavity, but nevertheless they are counted as if we had a dozen in the peritoneal cavity.

We use the figure-of-8 suture of silkworm-gut, just the same as we do in all our laparotomies. We are getting normal skin for this union in place of the cicatricial skin. The skin is closed with horsehair.

Let the record show further that after cleansing the field, the patient was placed in the upright position, and it was easy to invert the mucosa of the cecum. When this was completed, the margin of the mucous membrane of the cecum was entirely detached from the skin, and the margin of the cecum from the fibrous tissue of the muscular wall. As soon as that was fairly free, an entropion stitch of Pagenstecher linen was made, turning in the divided edge of the mucosa of the cecum. When this was completed, the cecum was freed from all its parietal attachments for a distance of two inches on the external side and perfectly freed on the internal side. Then two additional rows of Pagenstecher linen sutures were made, approximating the muscularis of the intestine. The fascial attachments and adherent omentum were entirely freed, so that the muscularis came into view.

When this was completed, a drainage-tube was placed external to the cecum and extending out into the connective tissue, that is, on the outer side of the cecum, and passing it to the pelvic or iliac floor. There is practically no tension on the skin. Horsehair has maintained the apposition. The drainage-tube was secured by a safety-pin, and with a silkworm-gut fixation suture. Bismuth subiodid will take up the secretion from the skin, if there is any, or it will form a cement with that secretion and seal the wound. It is better than collodion.

That is the uniform technic in the management of all external intestinal fistulas. First, get that bowel opening freed from the abdominal wall; second, close it; third, free the bowel from its attachment to the abdominal wall; fourth, put in the additional rows of suture that are necessary for the approximation; fifth, provide for drainage, if necessary. Suppose that was a cavity down in the pelvis and you have several loops of intestine coming in: First, open the peritoneum around the sinus, and then go wide to where you have free peritoneum and detach the bowel from the anterior abdominal wall, no matter whether it is the sigmoid or the upper portion of the rectum. Occasionally, in the upper portion of the rectum, for instance, where there is a suppurating dermoid that opens into the rectum and the fistula is located at that fold where the peritoneum turns forward from the rectum onto the uterus, where you cannot get sufficient freedom to make an inversion, you frequently have failure of union. Free it on all sides as far as you can, one-third of each side, and get a good approximation, and make a turning in, if possible. If you cannot make a turning in, put in stitches one after another, including the posterior surface of the uterus and suture the uterus down on the hole. I have done that a number of times with success. In that case always put in a rectal tube so that there is no tension during the process of repair. The tube should be kept in three weeks, so that there will be a free escape of gas and feces.

The worst class of cases encountered in this connection is the class associated with tuberculosis of the intestines. In that class of cases you always have danger of failure of union of the intestinal wall. If the fistula is large and the bowel is fixed and the tuberculosis is circumscribed, there is another route you can follow: You can detach the ileum from the colon, and reattach it to the colon higher up, entirely out of the tuberculous zone, making either an end-to-end or side-to-side anastomosis into the ascending colon, some distance above the cecum. Close the end of the cecum. Then you have simply a tuberculous cecum or a tuberculous pocket. Fatten up the patient until his tuberculosis is overcome, and then make an excision, but do not try to make the excision primarily, because if you do you will lose your patient. As a rule, you will get infection of the field, and, furthermore, you will not get union if you are close to the tuberculous zone.

[This patient made a rapid convalescence from the operation. The tube was removed within a week, and the wound closed promptly. The patient left the hospital in four weeks and reported a week later that she was feeling fine and was entirely well. Her doctor stated that her bowels were moving naturally, and that there was no evidence of any trouble in the right iliac fossa. Later word was received from patient that she was entirely well; no hernia; no weakness of abdominal wall.—Ed.]

MEDICOLEGAL RELATIONS OF PHYSICIAN AND PATIENT

DR. W. C. WOODWARD
Health Officer of the District of Columbia

Address delivered at Dr. Murphy's Clinic, October 21, 1912

DR. MURPHY: We are fortunate and favored in having with us today a man who has had institutional training and great experience. He has been health officer of Washington, D. C., for about eighteen years, and prior to that served as coroner. In addition he is professor of medical jurisprudence in Georgetown and George Washington Universities; in the former, professor of medical jurisprudence in the legal as well as in the medical department. He had his early training in medicine and later took up the study of law. I have the pleasure of presenting to you Dr. Woodward, of Washington, D. C., who will talk to you on the subject of the Medicolegal Relations of Physician and Patient.

DR. WOODWARD: *Gentlemen:* The subject I have selected to talk upon this morning is one to which too little importance is ordinarily attached in medical schools. The physician passes through his course in medicine, takes his diploma, obtains his State license, and begins to practise. That he has a definite legal responsibility to his patient does not often occur to him unless difficulty occurs in collecting some contested bill from a patient or from a deceased patient's estate. Or, possibly, he gets his first insight into the legal relations of patients and physicians through a malpractice suit instituted against him. A physician can evade the responsibilities of general practice. He may decline to take a case of obstetrics. He may limit his field of activities to surgery and decline to take medical cases. He can evade responsibility concerning practically everything in relation to the patient, except with respect to this one matter on which I will talk this morning: he cannot attend a patient and at the same time evade the legal responsibilities incident to such attendance. You may, however, search most of the statute books for statement of the legal relations of physician and patient, and you will find little or nothing concerning them, because they are regulated merely by the common law, as interpreted by a long line of court decisions.

The physician is under no obligation to treat any patient or to treat any class of patients. He may refuse to respond to any call that comes to his office, and for such refusal he is answerable only to his own conscience and to public opinion. When a human life is at stake in an emergency case, for instance, it is a physician's moral and ethical duty to humanity to care for the patient, even though the case be outside the line of his special field of work. But should he refuse to do so, even though the patient be injured by his refusal, he incurs no legal liability; the censure of the public and the pangs of his own conscience are the only penalties. Remember, then, that if you assume professional obligations to a patient you do so voluntarily. If you do not wish to assume them, you should not take the case. The obligations which the patient assumes to the physician are also generally voluntary on his part. There are, however, instances in which this is not the case. An infant or a lunatic does not voluntarily assume any obligations to the physician. Nor does a prisoner: when he is sick, the prison physician serves him whether the patient wants it or not; neither do men in the army and navy, who are served by the physicians in the service; nor does the man or woman who becomes unconscious on the street or in some public place, necessitating immediate medical aid. Aside from these few instances, the relation of the physician and the patient is always brought about by the voluntary act of the two parties.

The relation between physician and patient is commonly based upon what the law calls a contract, and a contract always signifies the meeting of the minds of the parties to a voluntary agreement and the assumption of mutual obligations by such parties. A physician may, however, have no contractual relations to his patient and yet be liable for any shortcoming on his part, whereby the patient is injured. Such, for instance, is the case of the physician treating a dispensary case, or the case of a physician who acts as an examiner for some public-service corporation. In these latter cases there is no contract between the physician and the patient or the person under examination, but if the physician injures him, the patient has a right to sue and to recover damages.

The contract between a physician and his patient may be any one of several well-established kinds. It may be written or it may be oral. It may be expressed in words or it may be implied from circumstances. The express contract is not necessarily a written contract; you can make an express contract by word of mouth just as well as you can make it in writing, the only difference being with respect to matter of proof. There are a few exceptions to this rule, which it is not necessary to discuss at length here, as ordinarily they do not occur in the practice of a physician. Generally speaking, if the parties agree by word of mouth or in writing each to do certain stated things, an express contract enforceable in law has been created. An implied contract, however, is one in which there is no such agreement expressed in words. In these cases the law steps in and says that the act and conduct of the patient and the physician imply an agreement on the part of each to do certain things even though, as a matter of fact, neither party ever gave the slightest thought to the terms of such an agreement.

A physician receives a call at his office and answers it. He finds a case of typhoid fever and continues to treat the patient. He may by law collect his bill, even though the patient never said a word about paying, and the patient may sue for malpractice, even though the physician never promised in as many words to treat him according to any standard. A contract may be in part expressed and in part implied. Contracts of this kind are frequently created with respect to operative work, the surgeon and the patient agreeing upon the amount that is to be paid for the surgeon's service, and leaving all other terms of the contract to the ordinary implications of law.

It may interest you to know that the expressed contract is commonly the contract of the quacks, although it is not necessarily so. The quack advertises that he will give a written contract that no money will be demanded if a cure is not made, and to many a poor man, or even to a rich man, who has spent much money with the reputable profession without obtaining relief the proposition is alluring. To go into the office of a physician and to receive treatment without any monetary obligation on your part unless you are cured seems attractive; most physicians do not guarantee so much. A careful examination of the guarantee which the quack will give, however, will frequently disclose a clause that protects the quack against any likelihood of suit.

I recall the case of a reputable, upright business man, who had increasing trouble in passing his urine. He had consulted some of the best physicians in the country, and they agreed that he was suffering from an enlarged prostate and that there was nothing to do for him except to operate. Dreading an operation, the man strayed into the office of a quack, who promised to cure him without an operation. The quack asked the patient, however, to give him as a guarantee of good faith promissory notes to the amount of the prospective bill in case of cure, the quack promising not to collect them until after the cure was effected. As you may well imagine, the patient was not cured, and when he demanded the return of his notes, he found that they had been negotiated and had passed into the hands of an innocent third party, for value, and without notice of the conditions under which they had been given to the quack. The patient then sought to recover from the quack the amount of the notes and took his guarantee to his attorney. But his attorney promptly discovered that the guarantee was adroitly worded so as to involve an admission on the part of the patient that the cause of his trouble was self-abuse. Rather than submit to the presentation of such a contract in court, the patient abandoned all efforts to recover the money out of which he had been swindled. Sometimes the contract which the quack enters into is in the form described above, in which the patient is technically made to admit that his troubles arose from self-abuse. In other cases the admissions on the part of the patients are of gonorrhea and of syphilis. Manifestly, the patient does not ordinarily care to go into court to enforce a contract in which he has technically made such an admission, even though he may be able to prove that the admission was fraudulently obtained and was not based on fact.

A physician can, if he so desires, lawfully contract with his patient that unless the patient is cured the physician will make no charge for his services, and if the physician does so he can collect no fee from his patient unless a cure is effected. There are, however, limits upon the contracts which a physician may lawfully make. He cannot lawfully contract to do an unlawful act. For instance, he cannot lawfully contract to murder, or to influence unduly a person in making a will, or to do a criminal abortion. If a physician should make such a contract and fail to carry it out, he could not be held responsible in court for the breach of the agreement. Moreover, a physician cannot take a case and relieve himself even by express agreement from the consequences of neglect or lack of ordinary knowledge and skill on his part. Such a contract is contrary to public policy; for certainly a physician should not undertake to care for a human life unless he is sufficiently careful and competent to do so. If he takes a human life into his hands, he must assume the burden; if he is unwilling to assume the burden, then he must not take the case.

The time when an express contract between a physician and his patient springs into existence is always sufficiently clear. The time when an implied contract is born is equally clear, although not so manifest on the face of the circumstances that give rise to it. A call comes to a physician or is sent to his office, and he or some responsible person employed by him promises that the physician will respond. Instantly a contract has been established between the physician and his patient, and if the patient relies on the physician's promise to respond to the call and is injured thereby, the physician is liable for any damage that may result. Or even without any promise by the physician or on his behalf he may respond to a call, and the very circumstance of him so responding gives rise to an implied contract between him and the patient from whom the call comes.

The duration of the physician's engagement to attend a particular case is a matter of considerable interest. It is not necessary that the physician should be summoned by a patient for each and every call; having accepted the call or having once visited the patient, the physician is under an implied contract bound to continue to attend the case. If it is not his intention to do so, he must give the patient definite notice that he desires to give up the case, and even then it is his duty to continue in attendance until another physician can be obtained or until his attendance is terminated by some act on the part of the patient.

There are several ways in which the physician's connection with a case may be terminated. The death of the patient terminates the contract, except in so far as relates to the subsequent liability on the part of the patient's estate or of those responsible for his care to pay the physician for his services. It is important for the physician to remember that death terminates his contract with the patient in so far as his professional services are concerned, for this shows him clearly that he has no control over the dead body and why a physician cannot lawfully do an autopsy without the express consent of the proper legal custodian of the corpse. The recovery of the patient will terminate the physician's obligations to attend the case quite as effectually as will the patient's death.

Cases occur, however, in which the patient does not die and does not recover—cases in which recovery is impossible, and in which the physician's services are in a certain sense of no medical value. Under such circumstances the contract on the part of the patient to pay for the physician's services must be regarded as at an end, unless the contract is extended by express agreement. Certainly it would not be right for a physician to continue in attendance on a case in which he knew he could render no benefit to the patient and to undertake to collect for visits made under such circumstances, unless the patient or some one responsible for the payment of his physician's bills clearly understands the circumstances. The physician may find that the patient or his family obtains comfort from his visits and desires him to continue in attendance even though his professional skill can avail nothing. Under such circumstances there is no legal or moral reason why his visits should not be continued, and if they are, the physician has a right to collect his fees; for a visit of this kind consumes the physician's time, which is his capital, quite as much as does a visit in which his professional skill and knowledge may be called into play with a likelihood of effecting a cure.

There is a third way in which the physician's relations to his patient may be terminated, and that is by the express wish of the patient. When this has been done the physician is, of course, under no further obligations to the patient. The patient does not have to give the physician any statement of any reason for dismissing him. On the other hand, however, if the physician desires to give up the case; if he feels, for instance, that he has lost the confidence of the patient and his family and that the strained relations are doing no one any good, he cannot give it up in any such summary way. There must always be due notice of a desire on his part to be relieved. By due notice is meant notice sufficient to allow the patient to obtain the services of some other physician. It has been questioned, indeed, whether a physician has a right to give up a case under any circumstances when the patient desires him to continue in attendance. The argument in favor of such a view is that a physician who is personally acquainted with the condition of the patient during the earlier days of the illness and who knows what lines of treatment have been followed and with what effect is in a much better position to care for the patient during the latter days of the case than any other physician possibly can be, and that under any and all circumstances the patient is harmed by the withdrawal of the physician who has cared for him first. It may well be supposed that the patient would have grave difficulty in proving that any actual harm had occurred by the physician's retirement, and in the absence of proof of injury he would be unable to get a verdict.

A contract between a physician and his patient always implies certain mutual obligations. It is necessary, however, to consider the obligations of the patient and those of the physician separately. To what extent, then, is a patient required by the ordinary contract between a physician and patient to cooperate with the attending physician? The patient, in the first place, is obliged to give the physician a fair history of the case; he must submit to any reasonable examination; he must follow all reasonable instructions received from his physician; and he must pay a proper fee. The obligations on the part of the physician are to care for the patient with due diligence, to bring to the service of the patient ordinary knowledge and skill, to use the best possible judgment, and to respect the confidence of the patient.

Time will not permit us to discuss at any length the obligations of the patient. Briefly, it is not necessary that the patient volunteer his history; the physician must suggest salient points and ask for information. The physician cannot expect his patient to volunteer or suggest that an examination of any kind be made; sometimes the patient does so, but more commonly he does not. If the physician's request for an examination is a reasonable one, and the patient does not give his consent, the patient alone is responsible for the consequences. The same is true with respect to the instructions that the patient should follow. Failure on the part of the physician to give instructions necessary for the proper treatment of the case renders the physician liable for any ill consequence that may ensue as a result thereof, but if the physician gives such instructions and the patient fails to follow them, the patient himself must bear the consequences. In the absence of an express agreement as to the amount the patient is to pay the obligation on the part of the patient is to pay as much as the services are worth, and this is determinable by the court in event of dispute. Under an express contract the physician can collect the amount that his patient may have agreed to pay, regardless of the actual value of the services, unless the patient is an infant, insane, or, in some jurisdictions, a married woman, or unless the agreement has been extorted by means of fraud or duress.

The obligations of the physician are, as I have already stated, reciprocal to those of the patient. In the first place, the physician must exercise due diligence. There are two ways of defining due diligence on the part of the physician: One is to say that it is diligence in proportion to the gravity of the case. The diligence given to a case of gonorrheal ophthalmia is not the diligence required for an ordinary catarrhal conjunctivitis. The diligence that is required in the amputation of a finger at the terminal phalanx would not suffice in a case of an amputation of a lower extremity at the hip-joint. Due diligence is defined also as the diligence which the average physician employs in similar cases. As diligence is with the average physician proportionate to the gravity of the case in hand, both definitions amount to the same thing.

The second of the obligations of the physicians is to serve his patient with ordinary knowledge and skill, and this is measured by the knowledge and skill of physicians at the same time in the same locality or in similar localities. The standard is not a very high standard. The physician does not need to be as learned as the best physician in his community, but he cannot be as incompetent as the poorest. So long as he keeps at or above the average, he does all that the law requires of him, and even though he may not cure his patient, or, in fact, though he may do actual injury, he cannot be called upon to respond in damages, at least not be called upon to do so on the basis of deficiency of knowledge or skill. In determining the legal requirements of knowledge and skill a physician is judged by the standards of the time and of the place in which he practises. A physician in Chicago could not call in a witness from a small rural town and prove that the Chicago practitioner was blameless because he had used the knowledge and skill of the average practitioner of the small town. Neither could he justify his practice by showing that it had been in general use years before or by showing that it might be in use some time in the future. A physician will be judged, too, according to the practice of the particular system of medicine with which he identifies himself; a physician of the homeopathic school will be judged by the standards of homeopathy, and a physician practising regular medicine will be judged accordingly. A physician who specializes in a certain line will be judged according to what he holds himself out to the public to be: not by the average of physicians generally, but by the average of specialists in his particular line.

Another criterion of the physician's work has reference to the exercise of judgment. He must use his very best judgment; not ordinary judgment, nor average judgment, but the very best judgment of which he is capable. And finally, but not least, among the physician's obligations comes his obligation to secrecy, or "confidentiality," as it has been termed, with respect to information obtained from patients professionally. It is greatly to the credit of the medical profession that it is so strict in the following of this rule of secrecy. So little is said in medical schools and in medical societies about it that it is a wonder that the rule is not violated often. The old Hippocratic oath, although not now formally administered, is yet commonly lived up to by the profession in so far as relates to its obligations to secrecy, and cases in which physicians have violated the confidences of patients are extremely rare. In every case it is the physician's duty to respect the confidences of his patient, even when testifying in court. The same rule holds true, of course, in reporting cases to medical societies or in publishing accounts of them in journals and in books. Under such circumstances, the identity of the patient does not interest the members of the society or the readers of the journal or books. They are interested in the case from a scientific standpoint only, and if the identity of the patient is not disclosed, the case can be published. Of course, if the patient consents to the disclosure of his identity, as he does when he voluntarily appears before a medical society or in a clinic, there can be no legal objection to the disclosure.

There are one or two matters in respect to the relations to physicians and patients which must be considered, which yet do not come under any of the foregoing heads. The first has reference to the physician's rights to examine his patient. An examination always requires consent on the part of the patient or on the part of some one who is authorized to give consent for him, but consent may either be expressed or implied. If a patient is asked to remove his clothing in order that an examination may be made or told to go into the next room so that the nurse may prepare her for examination, and if the patient does either of the things, the very act implies consent to the examination. The same is true of operations. Consent may be implied in the same way or it may be expressed. In connection with operations, however, it is necessary to consider very carefully the extent of the surgeon's rights to operate in any given case. The question arises not only as to whether consent has been given, but also as to what the consent was given for. The surgeon can lawfully do only so much as has been agreed upon, and it is always desirable to have full authority to use your discretion as a surgeon, obtaining it before the patient goes under the anesthetic, for it will be too late to think about the matter afterward.

An interesting case occurred a few years ago. A trained nurse had a diseased ovary, and knew that possibly both ovaries were diseased. According to her statement, she went to the operating-table with the understanding that if only one ovary was diseased it was to be removed, but that if both ovaries were diseased neither was to be removed; for she was engaged to be married and did not wish to be unsexed. The nurse was operated upon and both ovaries removed. Upon her recovery she sued the surgeon for damages. The surgeon claimed, however, that when the patient had given him the instructions which she claimed to have given, he replied that she would have to leave to his judgment what was to be done after the abdomen was opened and that to that reply she had acquiesced. Although the patient appeared to have no recollection of such acquiescence, the case was decided in favor of the surgeon. Unless the surgeon has the patient's authority to do whatever may be necessary, the surgeon must have the consent of some one authorized to answer on behalf of the patient before he can go beyond the operation agreed upon.

In a recent case a man was to be operated upon for a hernia on one side. When the patient was brought to the operating table under anesthesia, the surgeon found a hernia on the other side, in his judgment more dangerous than that upon which the operation was originally intended to be performed. The surgeon, exercising his own judgment in the matter, operated upon the more serious hernia, and the patient thereafter sued him for damages. In this case, however, the court took a common-sense view of the matter and rendered a verdict in favor of the surgeon. I think that it may safely be said that here the similarity of the operation proposed and the operation actually done influenced the verdict, and it would not be safe for any surgeon relying upon this case to do an operation radically different from that which was originally planned.

Another situation which the physician must be prepared to meet and to take the responsibility of if necessary is that of restraining his patient. Wherever restraint becomes necessary by reason of delirium or insanity or for any other reason, it is always better to place the burden of the actual restraint upon the family or upon those immediately responsible for the patient's care. Not that the physician has not the authority to exercise such restraint, for he can lawfully restrain a patient if such action is necessary to save a patient's life or that of others or to save him or them from serious bodily harm. If he does so, however, he may at least be made the defendant in a damage suit and be put to the trouble and expense of justifying his action. For this reason it is better that the burden be placed upon those who are called upon by natural ties to assume it and who are much less likely to be subjected to suit by a dissatisfied patient upon his recovery from the condition which made the restraint necessary.

All these obligations on the part of the physician and on the part of the patient would be of no use were not legal methods provided for enforcing them, or at least for procuring satisfaction for the injured party in event of their breach. It is impossible, this morning, to go at length into these methods. To do so would require a discussion of the legal responsibility of the patient and of various other persons, and possibly of corporations, for the payment of the physician's fees. It would be necessary, too, to discuss methods of enforcing payment in cases where payment could not otherwise be obtained. Malpractice, both civil and criminal, would also have to be discussed at length, and this would lead to a consideration of the matter of contributory negligence. Any of these subjects would afford ample matter for consideration for the full clinic period for the day, and we must, therefore, pass them by at the present time. I cannot close, however, without calling your attention to the fact that what I have discussed this morning have been merely the minimum limits to which the physician may descend and the minimum limits to which the patient may go without incurring legal liability. The physician who does not do at least as much as has been pointed out as his duty will be unable to collect his fees in a court of law. He may be made to answer in damages in a suit for malpractice. Conceivably he may even be fined and imprisoned in a prosecution growing out of criminal malpractice. The patient who does not do his part and who suffers injury may not only fail to get well, but he may, by reason of such contributory negligence, be unable to collect from the physician who attended him even for such damage as the physician's ignorance or neglect actually caused. I hope, however, that no one here is interested in determining how little he can do without subjecting himself to legal liability. I hope that no one here will be satisfied to win from his patient merely such a money payment for his services as the law authorizes. For if you would succeed in the practice of medicine it is necessary not that you do as little as the law requires of you, but that you do as much as your mental and physical strength will permit; that you be not merely as good as the average, but that you be as good as the best. And if when your patient has recovered, or even when your patient has died, the patient or his family feel that they have satisfied your just claims by a money payment; if they feel that they do not owe you any debt of gratitude and affection, you may know that you have not yet ascended to the higher planes of medical practice.

DR. MURPHY: I echo the sentiments of the gentlemen present in extending to Dr. Woodward our appreciation of his statement on the relations of the physician to his patient. We appreciate very much having him with us and giving us this instructive talk. I wish to thank you, doctor, for your kindness.

VISITING DOCTOR: Has the patient's physician the right to testify against the patient in a suit against a corporation?

DR. WOODWARD: If a physician is employed by a patient for the purpose of treating him for any injury or disease, he has no right to disclose any information to any corporation or to any party who is liable to be sued by the patient because of such injury or disease, unless the patient consents to such a disclosure. Whether he may be required to make such a disclosure in a court of law will depend upon the law of the particular jurisdiction in which the cases arises, and the laws of various jurisdictions regarding what are known as privileged communications vary so that it is impossible to say what will be required in any particular place without consulting the statutes and the decisions in that particular jurisdiction. If, however, a physician is not employed by the patient for the purpose of treating him, but is employed by a corporation which is liable to be sued or by a person who is liable to be sued, for the sole purpose of examining the patient and making a report, the physician clearly may make the examination and may report to the corporation or person who employed him what the result of the examination was.

TUBERCULOSIS OF KNEE: ARTHRODESIS

Treatment of Tuberculous Joints

HISTORY

Patient, a female aged forty-five years, comes to hospital on account of inability to use left knee-joint. Present trouble began in May, twenty-two years ago (1890), when she had pain in this joint when she walked a long distance or gave a sudden jerk to the joint. The pain was continuous, but not severe at first. It gradually became worse, and after one year and nine months she had to go to a hospital for three weeks. Before entering the hospital she had to use a cane. The week before this trouble began the patient had to remain home from school for a week with an ulcerated mouth and sore throat. Her knee was a little swollen when she entered the hospital. There it was painted with iodin, blistered with cantharides, and put up in a plaster cast for six weeks. After this cast was removed she wore a posterior metallic splint extending from calf to thigh for six months, and used a cane when going about. After this she wore an elastic knee-cap and got about without any support. When she came to this country twelve years ago the left leg was a little stiffer than the other, and she favored it a little when walking. She was able to get about without trouble until July, 1909, when she felt that her health was failing. She went to the country, but had to commence using a cane. Since then her knee has been getting gradually more painful and tender. She cannot flex her knee on account of the tenderness. No shooting pains at night. Is in good health otherwise. As a child had scarlet fever. No typhoid. Menses regular—twenty-eight-day type. No cough. Never had any cervical adenitis. Never injured joint to her knowledge. Tuberculin test gave patient nausea, malaise, headache, temperature 102.4° F., and increased pain in her knee.

COMMENTS AND OPERATION

Dr. Murphy: That began how many years ago?

The Intern: About twenty years ago.

Dr. Murphy: Was the knee swollen?

The Intern: Yes, doctor.

Dr. Murphy: "Slightly" swollen. That tells the story, because we must make a diagnosis on the original condition of twenty years ago. The clinical history is of the greatest possible significance in making a differential diagnosis of the lesion at the onset. Did you say the knee was "stiff"?

The Intern: No, doctor.

Dr. Murphy: No. That is important. If it were stiff or ankylosed, she would not need an operation. The fact that it is not ankylosed and is very painful is why she needs an operation. If she had a bony ankylosis, she would not need me. I will produce an ankylosis by removing the pathologic lesion that is there now and that has incapacitated her so long. There is considerable limitation of motion in the joint, and it is extremely painful and tender. An ankylosis after the lesion this woman had is one of the best results that can be secured.

What's the matter with her? What's the matter with her, judging by that history? She had a sore throat a week before she began to have pain in her joint, but when she got the pain in her joint it did not come on with a chill, it did not come on with a high temperature, it did not come on with an immediate incapacity, but it got a little more painful and a little more painful, until finally, at the end of six weeks, she was incapacitated. That is never the history of a metastatic arthritis of pyogenic origin. It is never the history of an epiphyseal or a metaphyseal osteitis of pyogenic origin. In both metastatic arthritis and metastatic osteitis of the ends of a bone the patient is incapacitated inside of forty-eight hours from the acute onset. The patient has a chill and an elevation of temperature. If the metastasis is in the synovial membrane, the joint is swollen. If

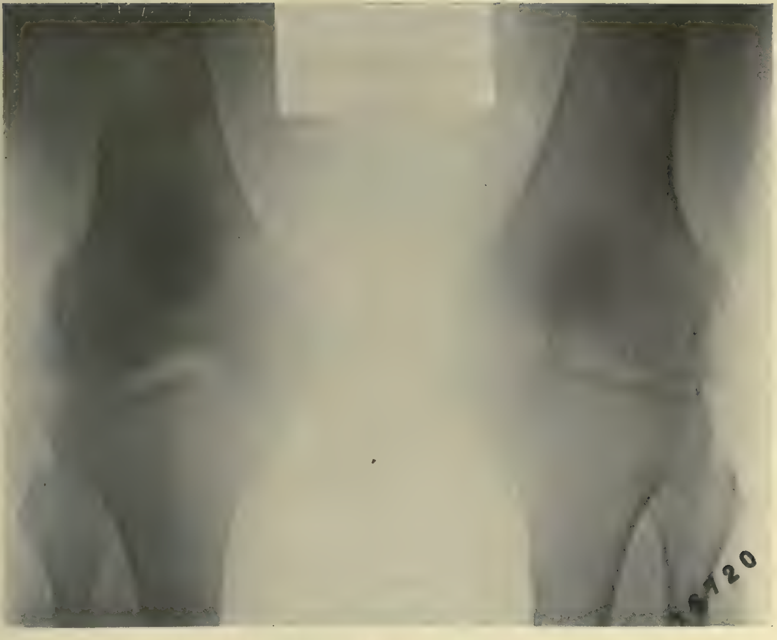


Fig. 18.—Right and left knee of case of tuberculous arthritis in which an arthrodesis was done. The internal condyle of the left femur (knee on right of picture) and the internal tuberosity were the seat of the disease. The right knee was normal.

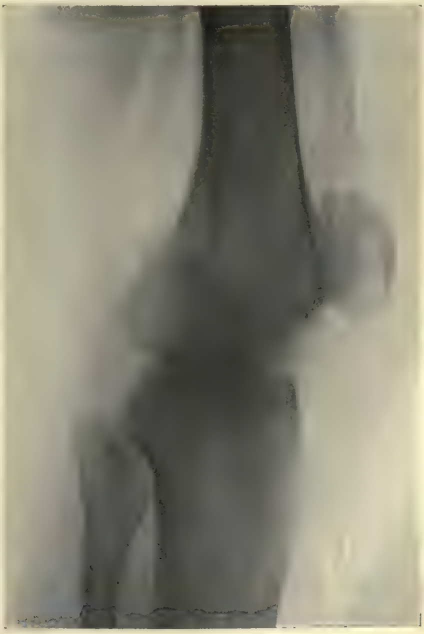


Fig. 19.—Lateral view, from inner side of leg, of the tuberculous knee-joint shown in the previous picture.

it is in the ends of the bone, above or below, the patient has intense pain, high temperature, the joint is not swollen, but it is incapacitated. What lesion is it that comes on in that slow, insidious way and that takes from three to seven weeks to incapacitate the patient, and causes limitation of motion? Tuberculosis. This is the typical history of a tuberculosis which repaired itself temporarily; whether it began in the bone or in the synovial membrane we do not know. We believe it began in the bone. Why? Because the doctor said the joint was "slightly" swollen. When it begins in the synovial membrane, which is rare in adults and common in children, the joint swells quite rapidly and to a considerable size and remains of that size for a long time. When a tuberculous focus appears first in the epiphysis and not in the synovial membrane, which is the common place in the knee, but always in a uniform place in the knee in adults—when it begins in the epiphysis, there is an effusion into the joint on account of the close proximity of the tuberculous infection to the synovial membrane, just the same as there may be a pleural effusion when there is an inflammatory condition in the lungs or a tuberculosis near the pleura, just the same as in cases of tuberculous epididymitis, there is an effusion into the tunica vaginalis, but in none of the three instances mentioned is there a tuberculosis of the skin or serous lined surfaces. It is the close proximity of the disease to these surfaces that causes the early effusion. That may entirely subside without there being any infection in the joint at all.

Just because of the fact that this patient had a sore throat or pharyngitis, which is a common cause of metastatic inflammation in the joint, we made a tuberculin test. We used five drops of dilution No. 2 of Lucius and Brunning's old tuberculin. That is our test dose in the average case, except in cases of acute enlarged lymph-nodes. If you use dilution No. 2 in these cases of acute enlarged glands, the reaction is too severe; the temperature rises to 105° to 105.5° F., and the patient has nausea and vomiting, and will be intensely sick. In the average case 5 minims of No. 2 is a justifiable dose. What is the reaction? That dose was given at 5 P. M. in this case. Why do we mention the hour? Because the temperature elevation of a tuberculin reaction appears, as a rule, between twenty and twenty-eight hours after the injection. If the patient's temperature rises to 103° to 104° F. within two or three hours after the injection, that is not a tuberculin reaction. That is caused by something else.

THE INTERN: The temperature was 102.4° F. twenty-two hours after the injection.

DR. MURPHY: That is the typical time, but a rather severe elevation of temperature. It usually rises to 101.3° or 101.4° F., but in this case it was 102.4° F. We had another case which gave a reaction of 102.8° on the same day. After one has used this test for a long time he becomes very familiar with it. Occasionally the temperature rises to 102.3° F. in two hours. We do not consider that a reaction at all. It is of no value.

Now, you see, she has no ankylosis. She has a limitation of motion, but no ankylosis. What do we wish to do here? Here is a woman who has been greatly reduced in her effectiveness and has suffered enormously for twenty years. What do we want to do? We cannot do an arthroplasty; we cannot get a new joint. What we can do and what we hope to do is to get a bony ankylosis. It is the best thing we can get for this woman, to give her a stiff knee.

A rubber tube constrictor has been placed high up around the thigh. The entire field of operation has been painted with 3.5 per cent. tincture of iodin. We are ready to begin the operation. A straight incision is made directly across the joint, just below the patella.

We are going to remove the patella. The edges of the incision are now held back with retractors to give us a good exposure of the joint. The capsule of the joint is now in view. These are the most difficult cases because of the pyogenic or saprophytic infection present with the tuberculosis. There is the ligamentum patellæ which I am dividing, and now the tuberculous exudate is welling out. You see she had an active reaction to the tuberculin test because she has an active tuberculous process here, with a mixed infection. Now we wish to remove all this tuberculous tissue. We will take out the patella and remove all the upper portion of this joint capsule with it. That exposes the joint. It is filled with pus and débris. The cartilages have been destroyed. The internal femoral condyle is eroded and devoid of cartilaginous covering. We do not wish to take off much of the articular ends of the femur and tibia, because this leg is to be straight, and if we take off much bone, the union will not be firm enough.

I am getting my bearings here as to how much bone needs to be removed. Here are the crucial ligaments. I wish to retain the posterior crucial ligament and displace it backward if I can, so as to aid in fixing the capsule. We do that regularly in our arthroplasties. I will make the angle of excision a little from before backward, as that gives the best subsequent result.

I am sawing off the articular surface of the femur where the patella was attached, so as to be sure and get out all the tuberculous débris. There is apparently no tuberculous lesion in the bones. It is normal bone as far as appearances go. If that is really true, then this tuberculosis was originally a synovial or a subsynovial tuberculosis. I am not so sure about that, however. The history did not indicate that. No, it was an osteal tuberculosis. There is a focus down deep in the inner tuberosity of the tibia, not connected with the surface. We will curet that out.

Let the records show that the disease began as an osseous tuberculosis in the internal tuberosity of the tibia, and that it extends down below the epiphyseal line. That is very rare. It is very rare to have a tuberculosis transgress the epiphyseal line. We have sawed off the articular ends of both femur and tibia, so as to get broad surfaces for approximation. We disinfect the field of operation thoroughly with a 5 per cent. phenol solution. We think it is the most effective means we have for protection against further infection in these cases. We are now ready to complete the operation by inserting an interosseous splint to aid in maintaining approximation and to favor bony union. We use magnesium for the interosseous splint. [A picture and description of this splint are shown in the June, 1912, number of the CLINICS.] Magnesium has the advantage of being dissolvable, and in tuberculous cases that is a disadvantage, because the absorption produces a gas which must be removed by aspiration from beneath the skin. The result of repeated aspirations is that a sinus occasionally forms between the two portions, which retards the definitive healing. It is a well-recognized truth in the management of tubercular lesions that, first, little or no catgut should be used as ligature material; second, phosphor-bronze and fine silver wire are the best ligature materials in these cases; third, after carbolic acid or other disinfectant solutions have been used to destroy the pathogenic microörganisms the cavity should be thoroughly washed out with salt solution and dressed dry. A little iodoform should be sprinkled over the surface to stimulate the connective-tissue formation and thus encapsulate or grow over any offending tubercle bacilli or tubercular disease that might remain. Magnesium has no special disadvantage in osteotomies, and acts as an immobilizer. It is the ordinary magnesium metal ribbon, about one inch wide and $\frac{1}{8}$ inch thick. We put it in in two different directions at right angles to each other, and in that way we get the desired immobility. Immobilization is a very important thing in all these cases. (In the more recent cases I have been using a silver or phosphor-bronze plate. The former is the better because it has antiseptic properties.) Two slots, an inch and a half in depth and at right angles to each other, are made in the ends of both bones. Into these slots the magnesium is placed—first into the tibia, and as the leg is slowly straightened with traction they are slid into the slots made for them in the femur. Now you see we have it complete. That prevents all motion, and this magnesium plate is absorbable and finally disappears, so that you never need to take it out. It lasts about eight to ten weeks. If there is much accumulation of hydrogen gas in the tissues, we aspirate and withdraw it just as we would fluid. As a rule, however, except in tuberculous cases, the gas is absorbed by the tissues.

Now we will suture the capsule with chromicized catgut. Our hope in that is to get what? It is to get an immobilization of the knee. The lateral capsule is the real support of the joint. Formerly we used the phosphor-bronze wire as a suture material here. It is a splendid material, except that it occasionally causes a sticking sensation, like a needle-prick, when it is exposed close to the skin, and it has been annoying in that way. We like it better than any kind of suture for this purpose. The fibrous capsule will be fixed firmly, so that it will not separate, but support the knee. The knee cannot rotate; it cannot move laterally on account of the method of inserting these magnesium plates. We rarely ever need to put on a ligature to stop bleeding. We have protected that wound from the skin by means of sterile towels all the time, so that our hands keep entirely free from contact with the skin.

We will now put in a few plain catgut sutures to unite the soft parts. The skin is united with horsehair. At the end of ten days we will commence to irradiate this knee with the Röntgen ray and stimulate the production of the encapsulation. A full-length plaster-of-Paris cast will be put on after we have dressed the wound with bismuth subiodid, collodion gauze, and 5 per cent. phenol gauze.

VISITING DOCTOR: Do you keep up the tuberculin treatment, doctor?

DR. MURPHY: All the time, after the wound gets over its acute reaction to the operation. That is, we commence to give tuberculin in fifteen days. Then we will continue that tuberculin treatment for probably a year. For therapeutic purposes we start with 5 drops of dilution No. 3, injecting it into the arm and repeating the injection every five to seven days, unless the reaction is too severe. We aim to get a temperature reaction of not over 99.6° F. The dose is increased two drops at a time until 20 drops is reached. Then we give two drops of No. 2, in the same manner as before, but increasing the dose only one drop at a time. When the dose again reaches 20 drops, we give one drop of dilution No. 1, increasing the dose one drop at a time. We have never given more than five drops at a time of dilution No. 1, because the patient has been thoroughly immunized when the treatment had progressed to that stage.

One of the greatest dangers in giving tuberculin is in giving too large a dose. A reaction temperature above 100° F., as a rule, indicates that the dose was too large, and that the opsonic index will be diminished rather than increased. I am surprised by the number of patients who come to me saying that they are taking tuberculin and that their reaction temperature is 101°, 102°, and even 103° F. These patients are being injured. There is no "average" dose. The individual reaction is the only criterion by which one gets accurately the proper administration of tuberculin as a therapeutic agent. Tuberculin is most powerful and efficient if given properly; it is highly destructive if improperly given. We always use in this clinic the Lucius and Brunning's preparation of old tuberculin.

Now let us go back and see how the pathologic findings correspond with the clinical history. She originally had a tuberculosis in the internal tuberosity of the tibia. How old is she now?

THE INTERN: Forty-five years.

DR. MURPHY: Twenty years ago she was twenty-five years old. The disease began at twenty-five. It would be exceptional to have it begin in the synovial membrane at twenty-five. It began in the bone, and slowly worked up into the joint. She was incapacitated temporarily in the beginning, with a little swelling of the knee, which later subsided. The tuberculosis encapsulated itself or healed by the formation of fibrous cicatricial tissue on the surface of the joint. She went on in fairly good condition until she had this manifestation of the further progress of the disease two years ago.

In analyzing the cases of bone and joint tuberculosis as they occur in this country we would say that we practically never see cases of tuberculosis that begin in the metaphysis, that is, that begin in the joint end of the shaft, either in children or in adults. I say practically never, but there are exceptions to that rule where the disease begins in the metaphysis. This occurs usually before ossification has taken place in the epiphysis. Then the disease begins in the joint end of the shaft. Mr. Stiles, of Edinburgh, in his articles on tuberculosis of the bone, said that in Scotland they very commonly have cases of tuberculosis of the shaft of the bone, and that they have them up to six, eight, and ten years of age. The interpretation which Mr. Stiles makes of that is that in Scotland it is more likely the bovine type of tuberculosis, because they drink so much milk and eat oatmeal—in fact, that is almost the exclusive diet at that age. The infection occurs in the metaphysis and not beyond the epiphyseal line toward the joint. The reason it does not occur in the epiphyseal line in children is that the epiphyseal line is a cartilaginous structure, and it is difficult or impossible to infect a cartilaginous structure with tuberculosis. Tuberculosis never primarily attacks the cartilage; neither does it ever primarily attack the fibrous portion of the capsule. Tuberculosis, therefore, in or near a joint occurs in one of three positions: in young children, very commonly in the synovial membrane; in older children, about ten years of age, in the epiphysis; and in adults it is almost always in the epiphyseal portion of the bone.

In the early stage of the disease in the metaphysis, the best operation is excision of the metaphysis, with the implantation of a portion of the tibia of the other leg after all evidence of the disease has subsided. There you have all the conditions favorable for the regeneration of bone. For instance, in the case of a child six years of age who has tuberculosis in the upper end of the tibial shaft cut into the periosteum, elevate it carefully from the bone, and cut the bone off above and below the disease, but always keep on the shaft side of the epiphyseal line. Just enucleate that bone out of the periosteum, and if there is no infection, immediately transplant a piece of bone from the opposite tibia, and the bone will be fully reproduced, but unless you put in some mechanical means of preserving the elongation and conformation of the bone during the process of regeneration, it will become twisted and deformed and be a poor limb, while if you put in a piece of tibia, it will keep that limb at its full length and bone regeneration will take place. We use the tibia because then we are guaranteed rapid reproduction, and the conformation of the limb is preserved from the beginning. It greatly stimulates the reproduction of bone in these cases. If there is infection present, do not transplant until the infection has ceased to give any evidence of activity.

The class of cases over which there is great controversy at the present time is synovial tuberculosis in the young. What will you do with a case of synovial tuberculosis—an epiphyseal tuberculosis of the hip-joint? How will that case be managed? These are the most difficult cases to handle, and it is only by an analysis of the changes of sentiment in the management and methods of treatment during the past twenty or thirty years that you can fairly balance the situation and the results obtained. You may remember that at the last meeting of the American Medical Association, and at the meeting preceding that, a surgeon advocated the immediate removal of the epiphyseal line, articular end and head of the bone, and the production of an immediate ankylosis in these cases. Let us analyze that treatment for a moment. I have been through the mill and I paid the price for learning. Thirty-four years ago exactly Dr. Christian Fenger, then a newcomer in Chicago, performed the first systematic excision of the joint as we did it this morning. I was then his intern in the Cook County Hospital, and became one of the early advocates of the removal of the tuberculous focus from the bone at an early date. What happens when you take off the head of the femur, including the epiphysis? The femur stops growing in length, and you get an ankylosis, but as the child grows up the disparity in the length of that limb and the other becomes greater and greater, and you have the patient in a worse condition than the tuberculosis could possibly have left him if you had done nothing. If you let it alone, the epiphysis will grow more, the head of the bone will finally absorb, there will be a shortening and sliding up of the shaft of the bone on the side of the ileum, there will be a fibrous or possibly a bony fixation—but he will have a limb in many cases as long as his other limb, in some cases much shorter. It is wrong to remove the epiphysis because it interferes with the growth of the bone in length.

We have to look at tuberculosis now from an entirely different standpoint than we did years ago. First, tuberculosis in children is much more readily curable than tuberculosis in adults. Children offer greater resistance to tuberculosis than adults. Tuberculous adenitis in children gets well, as a rule, with good food and plenty of fresh air. Tuberculous adenitis in adults does not get well in that way. I am speaking now of the lymph-nodes. I like to speak about conditions that we can see and feel. I do not want to speak about matters hidden in the uterus or away down in the esophagus. We want the things we can see when we speak of treatment and cure. In adults the disease is eradicated more readily with the aid of tuberculin and of hygienic conditions if there is no mixed infection. If there is a mixed infection and the gland is open, a cure is effected with tuberculin. We have not made a typical dissection in tuberculous cervical adenitis in this clinic in nearly seven years. It was our regular bread-and-butter work before that time. If we did not have two or three cases of tuberculous glands of the neck a week, we felt we were doing no business. These patients are all treated with tuberculin now, except the suppurating ones, and they all get well. [Dissection and microscopic examination of the gland show that it was tuberculous. The patient is since making rapid strides to recovery, although at first improvement was slow. —Ed.]

I have one case in the hospital now that came in while I was away on my vacation. The patient was treated here, but that case does not appear to be quite right to me. From the position of the gland it is a question in my mind whether this is not a case of Hodgkin's disease, which does not yield to tuberculin, but we are hoping that some day it will be determined that tuberculosis is at the bottom of it. These patients give a tuberculin reaction, but there is no effort at repair as in the case of a typical tuberculous gland.

These lesions in the joints occur in children more commonly in the synovial membrane, or if in the epiphysis, they rapidly rupture into the synovial cavity, so that you have a communication between the synovial cavity and the tuberculous focus in the bone; therefore you have a means of stimulating the process of repair in the joint and getting at the tuberculous pocket, which you do not have in adults. There is a local means of stimulating the repair in tuberculosis. One of the first things to result from tissue stimulation is the production of connective tissue. Tuberculosis is healed by fibrous encapsulation or surrounding the disease focus with connective tissue or by cofferdamming the lymph-spaces in the tissues. What can we do locally? We can aspirate the joint and inject it with an iodoform and glycerin emulsion, as we used to do years ago. It gave fairly good results because iodoform is a great stimulant of connective-tissue formation. We found in later years that formalin is a better stimulant of polymorpholeukocytosis. We used formalin and iodoform and glycerin for a number of years, and finally we dropped the iodoform, and we now use only formalin and glycerin. We inject a 2 per cent. solution, mixed at least twenty-four hours before it is used, and we always use a screw syringe, because the ordinary push syringe is not sufficiently powerful to force this solution through the needle into the joint. We introduce the needle into the joint very carefully, so as not to abrade the endothelial lining of the synovial membrane, and then make negative pressure. If the needle is in the joint, the synovial fluid will be drawn into the syringe. Then we make the injection. That has no effect at all on this class of cases, because you could not reach that focus in the bone with anything you inject into the knee-joint.

The next aid to repair is tuberculin. Tuberculin has a pronounced healing or repairing effect, not equaled in our experience by any other means of treating tuberculosis. I am speaking of tissue changes.

The next aid to repair is the stimulation one gets from the properly applied Roentgen ray. The Roentgen ray stimulates the production of connective tissue. That is how it cures carcinoma; that is why it acts so favorably in the repair of tuberculosis. Last summer we saw one of its most striking effects. I met a man, the most enthusiastic man on tuberculin next to myself, in Heidelberg—Dr. Wilms. He uses the Roentgen-ray and tuberculin. He has great confidence in the Roentgen ray, repeatedly applied and with a high-tension tube. Our treatments are given more often and with a low-tension tube. I believe that is a mistake on our part. Dr. Wilms showed us his result in joint after joint in which he followed his plan, and the process of repair is certainly striking. So much for the local treatment, that which stimulates the production of connective tissue, or the encapsulation of the tuberculosis by connective tissue and the immobilization of the joint by the cicatricial contraction that occurs.

What can we do next? In order to favor that process of cicatrization it is essential to get the parts immobilized. For a third of a century or more, since Sayre brought out his plaster-cast treatment, we immobilized tuberculous joints fairly well, but some one began substituting metallic and ambulatory splints, and all that type of apparatus, for the plaster-cast immobilizer. All ambulatory means of preventing intra-articular pressure are a delusion. You cannot prevent the pressure of the head of the femur against the acetabulum by any ambulatory splint that has so far been dreamed of or brought into execution. If you are going to immobilize it and lessen the destructive process, relieve the pressure in the joint. Extension of the Buck's type is the only means to prevent intra-articular pressure and friction during the process of repair. It is our plan to put these patients in bed, put on a Buck's extension with a sufficient weight attached to produce a separation of the articular surfaces, keeping the joint immobilized to prevent rotation, and give tuberculin. Tuberculin is the finest agent as an accessory. You remember the time when dear Dr. Fenger, whom we all loved and respected, would not operate for tuberculous peritonitis except on a sunny day, so that the sun could shine into the peritoneal cavity. We now know that the "sunshine" treatment of peritoneal lesions was entirely "moonshine."

So in the management of tuberculosis here we have a definite clean-cut plan of procedure, and we are getting better results than we ever did before. We are doing no primary excision of joints or bones in children at all. We believe the present endeavor to bring that method of treatment back into vogue again is wrong and will be abandoned. All one has to do is to analyze the results obtained when this treatment was first employed, and then cease to endeavor to revive this obsolete treatment. The tendency in every tissue in repairing tuberculosis is by self-limitation of the disease by encapsulation of the focus and healing. It needs every kind of stimulus that we have to produce this result. We put these patients first on tuberculin; then we use the Röntgen ray. Then there are the general feeding and fresh air; immobilization and elimination of contact in the joint. Many of these patients get perfectly well and remain well. That gives you an idea of our plan of treatment in this disease.

The skiagram in this case gives us no information at all as to the pathologic condition present. The tuberculin reaction was positive, and there was an active tuberculosis in the internal tuberosity of the tibia with a mixed infection.

Let the record show that there was a pocket of mixed infection between the internal tuberosity of the tibia and the internal condyle of the femur.

The plaster cast is now applied. It extends away up on the thigh. Under the cast we first placed a Gigli saw for the purpose of cutting the cast before the patient is taken out of the operating-room. We leave it open in place of closed. If the limb becomes greatly swollen, you can relax the cast as much as you wish.

PAGET'S DISEASE: AMPUTATION OF BREAST

HISTORY

Patient, a female, aged fifty-one years, comes to hospital on account of a cauliflower growth of left nipple and a large mass in the axilla.

Present trouble began August, 1910, as a little dry crust on nipple. This fell off and a little raw surface was left, which quickly crusted over again. This continued for four or five months, when a large crust formed and remained on about eight weeks. When this fell off a small growth on the nipple was found beneath it. This gradually and steadily grew larger, the whole nipple being involved in the growth, until its present size was reached. Seven months ago she found a small mass in her axilla. This was of about the size of a hazel-nut. This has also grown larger. No history of injury to breast; no pains; no previous inflammatory trouble; no pains in any of her bones.

Married at the age of twenty-seven; never was pregnant. Eighteen months ago patient was conscious of something high up in the vagina. After six months she went to a doctor, and he found a small polypus protruding from the cervix. This was cureted away.

Examination.—Nipple of left breast is a large, papillomatous mass, ulcerated on its surface, of the size of a silver dollar. The skin around the base of this mass is not involved, but there is a thickening beneath the skin around the base of the nipple. The remainder of the breast is free from any growth. Beneath the anterior axillary fold there is a large mass about the size of a medium-sized apple, stony hard, triangular in outline. The skin over it is adherent to it, but the mass is not adherent to the chest-wall. The supraclavicular glands are not enlarged. She has an enlarged thyroid.

COMMENTS AND OPERATIONS

Dr. Murphy: What's the matter with her?

The Intern: She has a malignant disease of the nipple with a metastasis in the axilla.

Dr. Murphy: What do you mean?

The Intern: It started as an eczematous condition of the nipple.

Dr. Murphy: It started first as a crust forming over the nipple, yes. What did the crust come from?

The Intern: It was an eczematous condition.

Dr. Murphy: No, it was not.

The Intern: It was a small papilloma in the nipple.

Dr. Murphy: It began in one of the lactiferous ducts. It is a case of Paget's disease. Paget's disease uniformly terminates in cancer of the breast,—and a severely malignant type of cancer at that,—and still Paget's disease is treated as an eczema, and the patient is permitted to go on to that awful condition that this patient is in now without being given relief. That is what I want to bring out, because these cases are the easily saved cases if seen early, before the lesion becomes malignant and metastases form. The lesion of the nipple is present often for years before there is any metastasis at all. They are the least malignant of any of the malignant neoplasms in the breast early in the stage of the disease.

When that little crust commences to form and when the patient consults the doctor because of that little crust, in place of giving her a salve for the eczema he should amputate the breast. Explain to her that that little crust is like the little crust on the lip of the smoker, that leads first to cancer and then to death. Tell her plainly that if she permits that to go on the chances are 100 per cent. that the case terminates in cancer, and in the enormous percentage of cases, when it develops into a tumor in the breast or axilla, it terminates fatally. We are responsible for many of these deaths, because these cases come to us early "for the eczema"; that crust is the secretion that comes out of the duct onto the surface, desiccates, and remains,

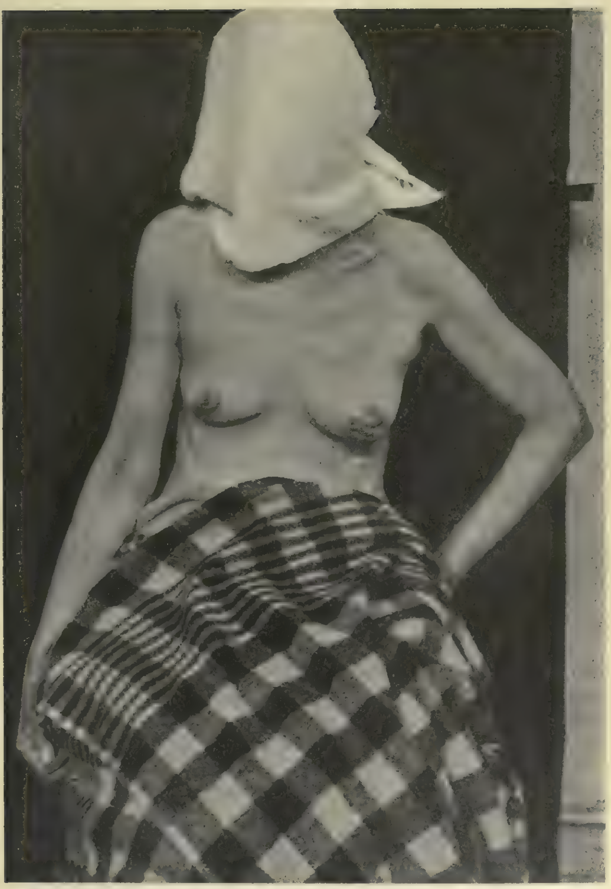


Fig. 20.—Paget's disease of left nipple, with a metastatic mass in anterior axillary fold.

and finally is a papilloma or a carcinoma. The epithelial edges of the nipple become red and fiery, like a condition known as "proud flesh," and that is the term the patient uses. She says, "Give me something to take the proud flesh off of the nipple," and if you know—and you should know—that that is a malignant neoplasm, you must not give her anything for her "proud flesh," or do anything unless she consents to an amputation of the breast. Be positive! let her go to another doctor,—and she will, after you have told her she is going to die,—but that prediction will haunt her, and she will go to five other doctors, and of the five, three will tell her it is malignant and she should have it taken out, and you will be vindicated. Sometimes the patient will only see one other doctor, and when she sees you the next time she will give you a haughty, frigid stare, because her doctor is curing her with a salve. Never mind! The point that should be emphasized is that she must not be permitted to sacrifice her life through an error in diagnosis. There is such a thing as an eczema in the nursing breast, but it is not this type—not the Paget's type.

There it is now, in the superlative degree. Look at that nipple! Look at that mass under her arm! The prognosis is bad. She will die a cancer death. She may live a little longer by having this operation, but she will die a cancer death just the same. We told her friends that would be the prognosis, whether she was operated on or not, although her life would be prolonged by the operation. This will be an extensive operation. The mass in the axilla is fixed to the skin, but not to the chest-wall. Therefore, we must take out that portion of the skin—in fact, do a radical operation.

We commence the incision just above the lower margin of the anterior axillary fold at about its center, and then pass inward over the chest to the sternum, encircling the breast and meeting the first portion of the incision at the base of the anterior axillary fold, near its inner angle. Then we dissect upward and outward toward the axilla, going downward on the chest-wall as far as the pectoralis major and minor. We cut these muscles at their inner margins close to their attachment to the ribs and reflect them outward onto the shoulder because we wish to utilize them later.

There is the aponeurosis of the pectoral muscle, which we will remove. I am going into the axilla first and clean that out from above downward before I touch the breast. That will eliminate the possibility of forcing cancer-cells into the general circulation. I thereby block the lymph-chain in the proximal direction. I carefully "wipe" the tissues off the vessels with a strip of gauze. The axillary artery is in plain view. See it pulsate!

I must remove all the glands and the gland-bearing tissue from this axilla, and I do so by "wiping," not cutting or tearing, it with scalpel or scissors.

We have everything in the axilla fully in view. I can show you now why I made a fatal prognosis in this case. There is an enlarged lymph-gland clear up under the clavicle. It lies right on the subclavian vein. I think there are other enlarged glands here. They may be in the mediastinum, where we cannot reach them from this field of attack. We will take out all the gland-bearing tissue. There are more glands here that I want to remove. I will remove all this fatty gland-bearing tissue right up to the clavicle, to make sure that we have removed all the glands in which metastasis may occur. That is very important.

I am back to the surface of the latissimus, *i. e.*, the posterior wall of the axilla. There is the nerve that supplies the latissimus dorsi—the long subscapular. It gives a faradic response. All this enormous amount of work is the story of the horseshoe nail. You remember the man who failed to put in the horseshoe nail and finally lost the horse? That's just what happened here. All that dissection had been made necessary because the warning of the little crust on the nipple was not heeded at the proper time. We will now remove the breast, together with the underlying pectoralis fascia, and also the fascia between the two pectoral muscles in which a lymph-node is occasionally situated.

We have done a most extensive operation here. We have removed all the gland-bearing tissue from the clavicle down to the level of the umbilicus. At the same time we have retained the pectoralis major muscle to fill in the axilla. We took out the aponeurosis of the pectoralis major and pectoralis minor. The long subscapular nerve is intact. The pectoral muscles are in view. The intercostal muscles are vibrating as she breathes; the axillary vein and artery, the nerves, everything up to the clavicle, are exposed. We are now ready to close up the wound.

The first thing I will do is to turn the pectoralis minor in to cover the first portion of the subclavian artery and to give such protection as was given by the tissue removed, as well as to diminish scar contraction and its effect in producing stasis of the venous circulation. We do not remove these pectoral muscles, first, because we believe it is unnecessary, and second, because we utilize them to fill in the axilla. We do remove all the pectoral fascia, the gland-bearing and lymphatic carrying tissue.

The pectoralis major will be brought up to form the floor of the axilla. It is sutured to the latissimus dorsi behind with a few catgut stitches. That fills in the axilla nicely. We will not have venous stasis, because that axilla is filled, and the contraction of the scar cannot compress the circulation or produce an edema or swelling of the arm or limit its motion. I had a patient here yesterday on whom I operated six or eight weeks ago. It is a pleasure to see the way she swings her arm. That is why we save the pectorals when we can and suture them into the axilla. When we cannot save them, we use the latissimus dorsi for the same purpose, swinging some of its fasciculi forward to fill in the axilla.

All that remains to be done is to close the wound without tension or as little as possible. We use silkworm-gut for that purpose. Horsehair approximates the skin incision. A small drain is placed in the lowermost angle of the wound.

Owing to the necessity of having to remove so much skin here it is impossible to close the wound without causing more tension than we believe is wise. Therefore we will close the wound as best we can with the view of doing a grafting operation later when the open area is covered over with granulations. There remains an area of, roughly, two inches wide and three inches long, which will have to be grafted. In ten days or two weeks the second operation will be done. A large dressing is put on with the arm by the side and a large gauze and cotton pad in the axilla.

[A Thiersch skin-graft of sufficient size to cover the denuded area was taken from the patient's thigh on the sixteenth day. It was spread out very carefully and sutured to the skin-edges with fine plain catgut. A wire screen cage was fashioned of the shape of a bee-hive, and placed over the field of operation, so that nothing in the way of gauze or cotton would come in contact with the graft. The cage was strapped to the chest with adhesive. The result was splendid. The graft fulfilled its function beautifully. The patient was discharged nine weeks after the first operation, with the wound completely healed, and with good use of the arm.—Ed.]

ACUTE APPENDICITIS

HISTORY

Patient, male, aged thirty-six, comes to hospital because of pain in the abdomen.

Present trouble began two days ago at 7 P. M. with a dull pain all through the abdomen. He took an enema and had a good bowel movement, but the pain kept getting worse. He could not find a comfortable position in bed. About 4 A. M. the pain began to localize in the lower right quadrant of the abdomen, where it remained all of that day. Was not nauseated nor did he vomit. About midnight of the second day he had a chill that lasted several minutes. Says he did not feel feverish. Has not taken any food since trouble began. About one month ago he had a similar attack, but no chill. It came on in the morning and lasted all day. That night he felt the pain in the right side, but it had disappeared the following morning, and he went to work. No other attacks. No fever at present time.

COMMENTS AND OPERATION

Dr. Murphy: You say the temperature was normal since coming to the hospital: not at all.

The Intern: It was subnormal.

Dr. Murphy: Yes; that's different. His trouble began about 7 P. M. the day before yesterday. All these things must be exact. It is a very important factor in the diagnosis of this case—one of the most important factors. Why is it of enormously greater value in this case to know that than it is in the average case? Let us go over this case. This case is not classic at all. It is very peculiar. He had the original attack and got over it, and was out and at work in two days. Then it looked like an attack of appendicitis, but it was more of the appendiceal retention type. He had an acute swelling of the appendix, which empties itself, and the patient has never had sufficient absorption of toxic material to give him an elevation of temperature. In all these years I have had only three cases of appendicitis without temperature and two within the last week. So far as we know, I can say this man did not have an elevation of temperature the night of the chill; at least no one took his temperature.

VISITING SURGEON: Doctor, may I ask you to repeat that last remark?

DR. MURPHY: In all these years I have had but three cases of appendicitis that did not have a temperature at some time in the attack. Remember, we often have a temperature within the first hours only in very bad cases; often no temperature at all when the abscess is as large as one's hat. That is usually accounted for by the fact that the temperature is the manifestation of the absorption of the products of infection and not the manifestation of their presence. See what a difference that is! It is a very great difference. We have for so long learned to associate temperature with pus that it is difficult for us to separate the two. Remember, temperature is not caused by the pus: it is the manifestation of the absorption of the pus. When there is no absorption of infective products there is no rise in temperature.

In this case no one took the temperature the night of the chill, but the doctor saw him yesterday morning and said he had no temperature the night before; the temperature was one-half a degree below normal. He had pain, but no vomiting. Whether he had nausea or not I could not learn. He is extremely sensitive over his right iliac zone—so sensitive that when I percussed it, not pressing on it, but simply percussing, he would scream. But his attack began with a chill, which is always a serious matter, because the chills occur so often when the appendix is becoming gangrenous. A chill occurs when the infective microörganisms or their products, or both, are taken up by the circulation in large quantities.

There is another point to which this history does not fit, and when the symptoms do not fit, then we always ask ourselves a great many questions, and we do a great many things we would otherwise not do. When I came in this morning I asked first for a report on his urine, because not infrequently a stone passing through the ureter gives rise to all the symptoms of appendicitis, without elevation of temperature. I immediately, therefore, asked for a report on the urine. The urine had not yet been examined, so I ordered the anesthesia postponed until we could get a report on the urine. If he has passed a urinary calculus, he will have blood in his urine this morning. He still has a temperature of 97.6° F.—below normal; but here is where we have an important sign appearing, a discrepancy between the leukocytosis and the temperature. The leukocytosis, if he had no infection, would be 7200, 7400, or 7500; after a meal it occasionally is more. He has not eaten anything, however, in forty-eight hours,—or rather sixty hours,—and his leukocytosis since last night has gone from 10,000 to 12,000. We have found that the leukocyte count is of value where the temperature is low, or nearly normal. I would not say normal, because we have not had it normal in any of the cases. When the temperature is 99°, 98.6°, or 100.5° F., we often find the leukocyte count to be 15,000 or 18,000. There it has very great value, while with my original syndrome in the order or "sequence of symptoms," as some one recently named it, that is, pain, nausea, and vomiting, local sensitiveness, and elevation of temperature, we expect and do find a leukocytosis present. We had a patient recently with a leukocyte count of 7000, and he had no elevation of temperature; his attack began at 4 o'clock in the morning; the doctor called me up by 'phone from Racine, and I advised him to bring his patient here at once. He brought the patient at once to Chicago, and I operated at 11 o'clock. Although he had no elevation of temperature and a leukocyte count of only 7000, he had acute sensitiveness in the appendiceal zone, and the appendix was full of pus and congested. The reason he did not have elevation of temperature was that the products of infection were not being absorbed into the circulation, but were leaking back into his caput coli, and the findings fitted perfectly to the symptoms.

This morning, with all our interpretations, as far as we know, this man should have an infection of his appendix, with edema and swelling of the appendix. Let me cite one case—and the more experience we have the more cautious we are about interpreting what else may be present, with the degree of absorption or with the degree of comparative normality of the appendix existing at the time of the operation. You can make a diagnosis of appendicitis in the earliest stages, but you had better not try to interpret the degree of pathologic disturbance in the appendix, because if you do you will suffer many humiliations. Make the diagnosis, operate on that diagnosis, and never mind the pathologic condition of the appendix. I will cite one case: I was called to the 'phone one evening at 8 o'clock by a doctor who stated he had a patient, a male, with a history of an acute, sudden, severe, intense pain in the abdomen which began in the morning at 8 o'clock. I was tired, and the case looked as though it would safely go over to the next day—about twenty-four hours in all since the onset of the pain. Never, or rarely, is there a perforation of the appendix or leakage of the contents of the appendix into the free peritoneal cavity inside of the first twenty-four hours except in cases of peritonitis from primary appendiceal perforation from stone. It was not a safe thing for me to postpone the operation. That night he had a pulse of 110 and a temperature of 104.5° F. I saw him at 8 o'clock the next morning and his pulse was 84 and temperature 99° F., and the man did not want an operation. I urged that he had appendicitis, and that he should have an operation notwithstanding his freedom from symptoms. He was a little sensitive on percussion. He accepted my advice and went to the hospital, and on opening his abdomen, what did we find? When that temperature went back to normal, I interpreted it as indicating that the appendix was draining back into his caput coli, but when I opened the abdomen, out popped a completely gangrenous appendix. Why did the symptoms subside? Because a dead appendix does not absorb nor does it cause pain. It was perfectly free in the abdomen, and it had died so quickly that it did not form adhesions to anything. It looked like a black rubber finger-cot filled with syrup. We lifted it out; the line of demarcation had completely formed, but suppuration had not taken place. That has made me more cautious about interpreting the sudden amelioration in symptoms. That appendix caused no elevation of temperature because with its death absorption ceased. Temperature elevation is the manifestation of absorption. That man had the most dangerous impending condition which he could have in his abdomen. I am cautious now in interpreting what I find, but the diagnoses are based on the same principles.

This man is very sensitive in the right iliac fossa. He had a chill. When they begin with a chill, I am much more apprehensive, because that shows that the type of pus that is present is the type that is rapidly disseminated through the lymphatics. This would cause the chill. The local infiltration will not be at all as great, but the danger to the whole body is greater. In operating on patients who have a chill, don't tear up the adhesions; if you find a pocket of pus, put in a tube and get out, because I know from that chill that we have a type of infection that the patient is not resisting, and he therefore must be kept from absorbing more than the smallest possible amount of infective products. You tide him over. One of the important things in medicine is to learn to individualize your cases and to learn just why certain things can or cannot be done, with the prime idea that you must get that appendix out at the earliest moment.

At the meeting in Budapest, 1909, where the subject was up for discussion before the International Congress, we had a very peculiar incident cited. A medical man came from Lausanne to talk on the subject of appendicitis, and one of the suggestions he made was that the abdomen should be punctured in the neighborhood of the appendix before operating to see if pus was present. That was only three years ago, and the irony of it all was that he came from the town where the first great work on surgery of the appendix was done by Kraft, also of Lausanne; he was the first surgeon to grasp the subject of appendicitis, and the first man who grasped the medical aspect of appendicitis was

Fitz, of Boston, and he insisted that it was a surgical disease. The point that Kraft made was that in perityphlitis, as the disease was known then, the pus was outside the appendix, came from the appendix to the outside, and should be liberated as in any other type of abscess; and then so many years after—twenty-one years—a man tells you that you should puncture the abdomen in the neighborhood of the appendix to see if pus is present, which shows that he never visited his neighbor's clinic or he would not have advocated that method of procedure. The statement which we made there was that every case of appendicitis that came for operation with pus outside of the appendix had in that fact incontrovertible proof that the case was inefficiently managed up to that time.

The diagnosis of appendicitis can be made better in the first twenty-four hours than later and the operation is easier.

Here is a case where you put a great big interrogation point after the diagnosis because it has had an exceptional course.

In making the incision for appendicitis we always go through the outer margin of the right rectus muscle. Divide the aponeurosis of the rectus, and displace all the muscle-fibers inward, so that the line of division of the fibers and the line of division of the aponeurosis are not overlying. That makes for a stronger abdominal wall afterward and makes the occurrence of hernia less likely.

The urine in this case was negative. The doctor reported on it while we were talking and it was negative.

The next is to pick up the peritoneum with two tissue forceps: but do not cut between the two forceps: cut on the incline made by the elevation. If you cut on the incline, you cannot cut the intestine at all, and you can cut it if you divide the peritoneum on the incline, between the forceps, provided the forceps hold the intestine under the peritoneum. After the peritoneal cavity is opened, the next thing is to endeavor to locate the appendix, and we do that by passing the finger outward along the anterior abdominal wall, then swinging it over the iliac vessels and up on the inner side of the iliac artery, where one hooks up the meso-ileum and elevates it or the colon into the incision. The meso-ileum is the most uniformly fixed tissue in the right iliac fossa. The meso-ileum is the fixed structure in this neighborhood. As I sweep my finger around under the colon, I bring out the appendix without adhesions. Isn't that a beauty? It is acutely infiltrated, distended, edematous, and fiery red. See the length of it! That is the time to pick them: pick them while they are green and don't wait until they are ripe. There is a case that in all my experience still left me in doubt as to the pathology. Now we come to the conclusion that the lesion is an appendiceal lesion. I do not know why he has not had certain symptoms, but this appendix shows us why he had the chill. See that thickened, infiltrated mesentery! That is why he had his chill. That is the best case we will have today as a life-saving proposition and as life-saving propaganda, because it shows you how these things have to be handled early in order to keep on the safe side, from the patient's standpoint.

We amputate the appendix in the usual manner, sterilizing the stump with 95 per cent. phenol. I want to show you how to cover that mesentery. I treat all these cases in the same way. There are both layers of the mesentery of the appendix. I will pass my needle through the one layer on the right, then on through the layer on the left, and then come back and make an overstitch. The overstitch first ligates the vessels, then comes through and approximates that peritoneum from both sides. Now I have a continuous circle of peritoneum for primary adhesion. We have from the beginning considered the opening made in the intestine by amputating the appendix the same as a bullet wound of the intestine, and we close it with exactly the same precautions.

We are back to the starting-point of that stitch. It is not pulled taut. It is going to be a little difficult to invert this appendix because of the thickness of the mucosa from the acute swelling, but still we will be able to invert it. We now put in the puckering-string suture in the base of the caput coli, the appendix stump being in the center of the loop of the suture.

You will have noticed how I pushed the infective contents of the appendix upward with the hemostat. I milked it up, and then ligated the appendix in the line where I crushed it with the hemostat. I want to keep the forceps on the appendix until we make a smear and a culture of its contents, and also from its wall. Then I amputated it and touched the stump with 95 per cent. phenol. I do not neutralize it, because I am going to turn it in, and it will take care of itself. The next step is to pull taut the purse-string suture and invert the appendix. The doctor inverts that. The next step is to put in a top stitch. It is put in as a Lembert stitch. That puckering-string is also a Lembert stitch.

Now we have closed the end of the intestine. We will put an extra ligature on the mesentery, because it is necessitated by this little spurting vessel.

Now we will look over the field. It is perfectly dry and clean. The next step is the closure of the peritoneal opening with catgut, and that closure is always done in a definite way, no matter what the operation is for. We produce an ectropion of the divided edge of the peritoneum, turning the cut edge out into the wound in the abdominal wall. That avoids peritoneal or intestinal adhesions at the line of operation, and that is a very important factor in every abdominal operation. Adhesions produce symptoms of distress and discomfort that the patient is entitled to be without, and that we can avoid, as we have for many years, by the use of this method of suture.

Each stitch is put in beneath the edge, from the under side, so as to turn the cut edge out, and the last stitch is put in as a puckering-string. That closes the peritoneal cavity. Now, we locate the lower and the upper angles of the wound by catching the aponeurosis of the rectus with hemostats. You can see that aponeurosis standing out there. Then we make an edge-to-edge suture union of that. Just unite it edge to edge. That gives us the best type of closure—an edge-to-edge union of similar histologic elements, a law which we laid down when we wrote our first article on the use of the button. It lessens the danger of stretching, and, on the other hand, of contracting, which has the danger of annular cicatrices that cause stenosis.

I want the intern to read that history again, so that we can check it up with the pathologic findings.

[Intern reads.]

Dr. Murphy: The patient does not just get the question of nausea. He is a German, and I asked him if he had "Übel." He was not so certain about that, but he did not have a marked tendency to vomit; that was certain.

(To intern): The Labor Day attack was not associated with a chill?

The Intern: No, doctor.

Dr. Murphy: Put that in the history. Then it was not a "similar attack." The chill makes a very great difference. His urine was examined and found negative; no blood. That was after riding three or four miles to the hospital.

Next we insert four or five silkworm-gut figure-of-8 sutures, which will approximate all the structures and obliterate dead spaces.

We close the skin with horsehair and then we will show you how we put on the gauze splint to prevent the figure-of-8 sutures from doing mischief by cutting through the skin. The gauze is laid folded on the wound, and the sutures are tied over it. The collodion gauze is put on over that and seals the wound.

Now let us go over the diagnosis and the things that are absent. We laid down a syndrome of symptoms for appendicitis—pain, nausea, vomiting, local sensitiveness, elevation of temperature, and leukocytosis. We never interpret a case as appendicitis that has temperature preceding the pain. I impress on you the significance of the order of the symptoms. One of my patients, many years ago, was sick from Friday, feeling badly the week before, but worked every day; but on Saturday night, after work, she went to see a doctor. She was a girl about twenty years of age. The doctor examined her, took her temperature, and it was 102.5° F. on Saturday night. She had no pain at all. Tuesday afternoon at 3 o'clock I saw her, and she had a temperature of 103.5° F. and had pain in the right iliac fossa that day; she had nausea and vomiting that day, and local sensitiveness with the elevation of temperature. It looked like a clean-cut case of appendicitis to me with my then rather limited experience of 100 or 200 cases. But she had a temperature before she had the pain. Pain in the appendix is a manifestation of the swelling of the infected mucosa, and its strangulation by the inelastic fibers and muscular coats and peritoneum of the appendix wall. Nausea and vomiting are merely reflex evidences of that strangulation. Elevation of temperature is the manifestation of the absorption of the infective products. You cannot have that absorption from the infection before the swelling appears. That tells the story. We operated on that girl, opened the appendix, which was very much distended, and found an ulcer of its mucosa. There were also innumerable typhoid ulcers all over the small intestine. The ulcer in the appendix was a typical typhoid ulcer. Since that time we have had 9 similar cases; the first 3 were operated—this was before the days of the Widal test; 6 were not operated. All the patients got well,—those that were operated and those that were not operated,—but we put an additional tax on the patients by operating. It is not advisable to operate on patients having typhoid ulcers in the appendix, notwithstanding there are a number of cases in the literature where the typhoid ulcers have perforated the appendix, for the number is not great enough to add the danger of an operation to an already serious disease.

In all the other cases I was able to get a history of temperature preceding the pain. The moment we obtain this fact that settles the diagnosis so far as simple appendicitis is concerned, and I cite that exceptional case because it puts us on our guard when the course is atypical, as it was in this particular case.

That is the method in which this wound is closed. First unite the peritoneum, turning the edges out. Next unite the aponeurosis, edge to edge; then put in a figure-of-8 silkworm-gut stitch. The lower loop has the aponeurosis of the rectus and the upper has all the space between the rectus and skin. After that is completed and the skin is closed with horsehair, we put over that a fold of gauze. The lower loop of the figure-of-8 stitch, when it is tied, pulls the aponeurosis close to the skin and obliterates the dead space. In that way we get better union and have a nicer course for the patient. This stitch is not original with us. It was used by Dr. Ill, of Newark, N. J., but he included all of the abdominal wall in the figure-of-8.

Over this we put on the usual abdominal dressing, which is held in place with adhesive straps and a binder.

Let the record show that Sister Victorine reports the sponges counted and correct. The sister is responsible for the sponges, and the intern is responsible for putting it on the record. If we are doing a laparotomy we always ask whether the sponges are all out.

ABSCESS OF NECK

HISTORY

Patient, male, aged twenty-five, comes to hospital on account of swelling over the right wing of the thyroid cartilage.

Patient first noticed the swelling three weeks ago; while undressing one night he found that his collar-band was tight. The following morning he noticed the swelling. In May, 1911, he was throttled, and his neck was severely bruised and scratched in the thyroid region. It was tender and sensitive for ten days, but this condition gradually subsided.

Examination shows a swelling to the right of the median line of the neck, about the size of a pigeon's egg, extending from the hyoid bone down to the cricoid cartilage. Mass is fluctuating, but quite tense. About a week ago it was aspirated and a small quantity of creamy pus was removed. Patient has had no symptoms of an acute infection, no carious teeth or enlarged glands. Great cornu of hyoid bone is broken on right side.

COMMENTS AND OPERATION

Dr. Murphy: This case is an interesting one. It involves peculiarities that are not at all common. The history of the case was that he had a trauma seventeen months ago, and that his neck did not begin to swell until about one month ago. One of the first things one thinks of in connection with a trauma in this region is fracture of the thyroid cartilage. He had no temperature; no chill; no evidence that he expectorated blood at the time of the injury; no evidence that his cartilage was fractured through the mucosa to give him the infection.

The next thing you think of is the thyroglossal duct, and that this may be a cyst of the dermoid type. After you have eliminated these two conditions you consider aberrant thyroids, namely, those that are situated along the normal anatomic tract of the thyroglossal duct in the embryo. You know that occasionally remnants of the thyroglossal duct are arrested all the way down this tract; that some remain in the middle, some are displaced to the side and remain away up under the tongue, and we occasionally have developed, as a sequence of that, first, a dermoid cyst lined with epithelial cells; second, an enlargement of the duct extending down from the foramen cæcum. Often it is lined with columnar epithelial cells from the mouth—a mucous lining. Third, a cyst shut off both above and below—a typical dermoid. That is a remnant of the thyroglossal duct and it is lined with epithelial cells. Fourth, there are developed in this tract the various changes that occur in the thyroid when it is in the normal position, namely, a sublingual goiter, a hyoid goiter, and a goiter anterior to the larynx. The sublingual goiter is the most common type. Occasionally, when that undergoes its goitrous change, only a portion of the thyroid remains. You enucleate that gland, and to your horror and surprise, in five or six weeks the patient commences to develop a myxedema. I will never forget my first shock in that type of case. The goiter developed beneath the tongue. We enucleated the mass, and when I cut into it I saw it was thyroid tissue. Then I commenced to look for the remaining thyroid and could not find any. We kept her in the hospital for a while until she commenced to develop an edema of the lips and of the hands. She got a typical myxedema, showing that I had taken out the only thyroid tissue she had. We put her on desiccated sheep's thyroid. All her symptoms disappeared at the end of six months; then we skipped a week, and then a little longer, and in fourteen months she was completely off the thyroid medication; either she had developed some thyroid cells somewhere, or she had some compensating cells. However, all the symptoms disappeared.

When this patient came here we had a significant connection element, the trauma, which made us fear that it was a malignant sequence of the trauma. This condition began suddenly and without any apparent connection with the trauma. We aspirated it the day he entered the hospital, about a week ago, and the fluid contained a few lymphocytes, a large number of polymorphonuclear leukocytes; no bacteria were found in the smear and no evidence of tuberculosis. It is, therefore, an acute infection, possibly through an involuted portion of the mucosa that resulted from a fracture of the larynx by the trauma. There is no connection with the larynx in this case because there is no discharge into his throat at all. The suppurative focus is apparently completely encapsulated.

We will freeze the skin with ethyl chlorid, which is the ideal anesthetic for these cases. Then we will expose the enlargement by a small transverse incision. This is suspicious of a dermoid condition. This infection is probably simply a coincidence in connection with the trauma. We will completely enucleate the sac, for such it is. It is filled with a gelatinous material which makes me fear that it is a dermoid, although the microscopic findings do not warrant that conclusion.

We will put in a small rubber tube drain for a few days or a week. The bleeding has stopped; only a few points of hemorrhage had to be ligated. We will close the wound with three or four horsehair sutures and put on a plain dressing.

[The drainage-tube was removed on the fifth day. The wound healed rapidly, the patient leaving the hospital on the fourteenth day.—Ed.]

BROAD LIGAMENT ABSCESS; PYOSALPINX

HISTORY

Patient, female, aged thirty-five, comes to hospital on account of pain in the left and the right iliac region.

Present trouble dates from last July (five months ago), when the patient had a miscarriage during the second month of pregnancy. Preceding the miscarriage she had slight uterine hemorrhage with painful contractions. Two days after the onset of these symptoms the uterus was cureted. That night the patient had a temperature of 103° F., which continued with slight fluctuations for two weeks. The day following the curetment the patient had severe pain in the right iliac region, accompanied by marked rigidity and tenderness and vomiting, which continued at irregular intervals for four weeks. One week after the onset of pain in the right side pain was also present in left side. This was followed the next day by general abdominal pain and rigidity. No vaginal discharge followed the curetment. During the second week of her illness the patient had one or two blood-tinged bowel movements. At the end of six weeks all her symptoms had subsided, but soreness was present over left and right iliac regions and occasionally in the lumbar region. Pain now seemed to be located in the hips and lumbar region and was aggravated by walking. The pain has been continuous since the illness. For the past eight or nine days patient complains of sudden, sharp pains across abdomen, which persist only a few seconds. Has vomited every day for the past eight days. Immediately after eating, usually the evening meal, the patient feels nauseated and vomits. No pain at the time. Complains of two tender areas—one in right and one in the left iliac region, the latter being the more severe.

COMMENTS AND OPERATION

Dr. Murphy: What is the matter with her?

Intern: Following that miscarriage she had a pelvic abscess.

Dr. Murphy: What do you mean by that?

Intern: She had an infection of the uterus, and the infection traveled from the uterus to the tubes and then into the peritoneum. That accounted for the temperature, pain, and rigidity.

Dr. Murphy: Why do you think so, doctor?

Intern: If she had a vaginal discharge before the miscarriage, I would think it was a Neisserian infection.

Dr. Murphy: I think there was pus in the tubes, on account of the presence of tenderness. When did that begin?

Intern: Last July.

Dr. Murphy: Following the miscarriage?

Intern: Yes.

Dr. Murphy: She had pain where?

Intern: First in the right, then in the left, iliac region. The pain in the right side disappeared when the pain in the left side began. She had abdominal rigidity and temperature.

Dr. Murphy: She had fever. She first had a chill and then a temperature and then pain. What is the matter with her?

Intern: She had an infection in her pelvis.

Dr. Murphy: That is very fine. You might as well say when a patient had a purulent synovitis of the knee that she had an infection in the lower extremity. That is too indefinite.

Intern: She had an infection in——

Dr. Murphy: Wait a minute. She had pain in the right iliac region and it disappeared promptly from the right side and began in the left. What is the matter with her? First, she had a chill, then fever, and then pain. The pain shifted from the right to the left side. What is the matter with her?

Intern: She has a tubal infection.

Dr. Murphy: It is an infection of the uterine surface and goes where?

Intern: Into the Fallopian tubes.

Dr. Murphy: No, it does not. It goes into the Fallopian tubes in a Neisserian infection, but rarely from a post-abortion infection.

INTERN: It travels through the lymphatics of the uterus.

DR. MURPHY: To where?

INTERN: To the peritoneum.

DR. MURPHY: Go on—the lymphatics going from the uterus to the peritoneum——

INTERN: It goes to the muscles.

DR. MURPHY: No. What is the matter with her? If you have an infection of the finger, where does it go—to the muscles?

INTERN: It goes to the lymphatics and then to——

DR. MURPHY: To the cellular tissue in the forearm. In this case where does it go? It goes to the cellular tissue of the broad ligament and then what happens?

INTERN: An infection of the cellular tissue of the broad ligament, and that may go on and form an abscess.

DR. MURPHY: That is a typical history of a pelvic cellular infection from a curetment following a miscarriage. The infection was on the surface of the uterus, passed through the lymphatics in the cervicocorporeal portion of the uterus, into the cellular tissue of the broad ligament, produced edema, swelling, and probably necrosis and abscess formation. I can swing the uterus to the right side, but not to the left side. I put in an aspirating needle through the vagina yesterday and drew out pus. How am I going to take out that pus center? How would you treat it, doctor?

INTERN: I would go through the peritoneum into the broad ligament.

DR. MURPHY: Then she would get a peritonitis and die.

INTERN: I would put in a vaginal drain from below.

DR. MURPHY: How would you do that?

INTERN: Put it into the culdesac.

DR. MURPHY: What would you do with the round ligament?

INTERN: I would drain it from beneath.

DR. MURPHY: What happens when you drain a pus-tube?

INTERN: It fills up again.

Dr. Murphy: What happens when you drain an abscess in the broad ligament?

Intern: As soon as you take the tube out it fills up again.

Dr. Murphy: Why?

Intern: Because there is some of the infection left behind.

Dr. Murphy: What happens when you drain a phlegmon of the thigh?

Intern: It is healed.

Dr. Murphy: What happens in the broad ligament?

Intern: It contains connective tissue and——

Dr. Murphy: It heals too. What happens when you drain a tube?

Intern: It refills.

Dr. Murphy: Why?

Intern: Because it has a mucous lining.

Dr. Murphy: And its fimbriated end is closed and its uterine end is usually closed, and with an infected mucous membrane in between, what must happen? A refilling of the tube with pus. I want you to say that this is an abscess in the cellular tissue of the broad ligament. It does not require a laparotomy; it needs to be opened from below, if you can do so properly. But it is in the broad ligament, which is ordinary connective tissue, and all we need to do is to drain. When you have an abscess in the Douglas pouch all it needs is drainage. When you have a tubal infection, a drain does not do any good because it is an infected mucous-lined surface. That is what I want to bring out. It is not a pelvic infection. Its clinical course is just as clean cut as is that of an infection of the thigh.

OPERATION

(the following day)

Dr. Murphy: Did she have a chill when she had that attack?

Intern: No, doctor. She had fever from the second day.

Dr. Murphy: Did she have a rigor the night preceding the onset?

Intern: She does not remember. She cannot remember ever having had a chill, but she remembers the fever.

Dr. Murphy: She must have had a chill. That type always begins with a chill. You will see that she had fever on the night of the curetment—not on the second day after, but on the night of the curetment. From that curetment, what else should she have had?

Intern: Temperature that night and——

Dr. Murphy: Temperature that night and pain first on the right side and then on the left side. The pain in the right side disappeared when the pain in the left side began. What is the matter with her?

Intern: She has a pelvic cellulitis.

Dr. Murphy: The doctor was over this case yesterday and he has a better grasp of the subject today than he had then. Let me see if you know what caused the pelvic cellulitis.

Intern: The miscarriage first and then the curetment and——

Dr. Murphy: That leaves the uterine surface raw and open to infection. That is just what the curetment does. When you scrape the surface of the uterus, you open up the lymph-spaces. When you open the lymphatics into the broad ligament, one of two things may occur in the broad ligament: First, great edema and swelling. The edema goes through a course of a number of weeks and an abscess forms in the ligament; second, the infection is carried into the broad ligament, and an abscess is formed in the cellular tissue of the broad ligament, accompanied by continued fever persisting for weeks.

When I was in the Cook County Hospital we had a great number of these cases as a result of curetments and abortions. When you examine the uterus in these cases you will find you cannot move it in any direction. The Germans say it is "eingemauert"—set in masonry—it is so immovable. It is just the opposite in this case. There is a mass in the left side, well back. The reason I am going to go through the abdomen is that I do not want to hazard the ureter. We open the abdomen in the usual manner, going through the right rectus muscle. There is the tube. This differs from the usual type of infection of the tubes. She may have a tubal infection connected with this, but it does not feel like a tubal infection. In tubal infections the infection occasionally passes from the uterus into the other tube. It may endeavor to go from the tube into the peritoneum by rupture of the tube or patency of the fimbriated end, and occasionally from the tube it passes into the Douglas pouch, but that is very rare, because when you get an infection in the tube it usually seals its end and retains the infection, so that it becomes a pus infection of the tube only. The infection does not drain back into the uterus and it becomes a pyosalpinx. That is just the opposite of what takes place in a Neisserian or gonococcus infection. In the staphylococcus and streptococcus infections the mucous infiltration temporarily blocks the tube at the uterine end without developing a stricture. With the Neisserian infections there is temporary blocking with finally a stricture developing at the uterine end of the tube, just as it does in the male urethra.

In examining the patient yesterday I looked for this infected zone from below. I went behind the insertion of the ureter and succeeded in aspirating some pus, thus taking off the tension. A large quantity of pus was withdrawn. Now, when you open the abdomen in these cases, what are you going to do? It depends on what you find. If the infection is in the broad ligament, the abscess is in the cellular tissue and all you have to do is to put in a drain. If it has passed into the tube, then you cannot drain. If it has gone through the tube and the tube rests on an ovarian follicle, it can infect it, and you can have a typical tubo-ovarian abscess.

The abscess appears to be situated anteriorly. I shall try to get into the Douglas pouch without opening any adhesions or fixations that have formed. The abscess is in the left broad ligament. There is the tube. There is also an infiltration in this uterus, but there is the abscess in the broad ligament. I will see what disposition I will make of this tube.

There is no history of a discharge of pus through the bowel. I am endeavoring to get hold of the fundus of this uterus. Here is the fundus of the uterus. This tube is infiltrated. Whether it is an infection or merely an agglutination I am unable to tell until I go in farther.

There is the appendix adherent to the surrounding organs. This tube will have to come out because it is wrecked as a tube. The fundus of the uterus comes right up into the field. I would say that this was a colon bacillus infection, from the appearance of it. We will have the pus examined for Neisserian infection.

Now we come to a more difficult situation—the tubal involvement. There is the wall of the pelvic abscess. I do not intend to take that out. Now we will commence to enucleate this tube. This tube has pus in it, and it has an offensive odor that suggests the colon bacillus infection. The other tube is also infected.

Here is the abscess in the broad ligament. Now we will clean it up. There is the end of the tube. Swinging it out we come on to its abscessed wall. See! we are right up into the cornu of the uterus. We will remove both tubes. I am opening the abscess at its upper pole, and then we will put in a drain. You see why I am not trying to take "a sac" out of this broad ligament. We do not take out the sac, but treat it just as we do an abscess in the thigh.

Let the record show that she had an abscess down in the pelvis and that the pus had an offensive, colon bacillus odor. Neither of the ovaries was removed, but both tubes were removed. Both tubes were infected. There was a double abscess in the left broad ligament, no abscess in the right broad ligament. With its clinical course we are unable to say whether these infections in the tubes, one with a colon bacillus odor and one without, are of Neisserian origin or not, but I would say they were not.

Let the records show that Sister Victorine reports the sponges counted and correct.

That is a very interesting case in its clinical findings. If she had not been cureted, she would not have had that broad-ligament trouble.

We close the abdomen, securing the drain with a silkworm-gut fixation suture. The peritoneum is closed with catgut, its edges being everted to prevent the occurrence of peritoneal adhesions. The rectus fascia is likewise sutured with catgut. Silkworm-gut figure-of-8 sutures and horsehair skin sutures complete the closure. The wound is dusted with bismuth subiodid powder and sealed with collodion gauze. The usual dressing is put on.

[At the time of reporting this case the patient was still in the hospital. The wound was draining well. The patient was comfortable. No temperature.—Ed.]

CEREBRAL ADHESIONS (DECOMPRESSION)

HISTORY

Patient, a male, aged thirty-four, comes to hospital on account of difficulty in speaking and numbness in right hand and leg. Present trouble dates from an injury twelve years ago, when he was struck in left parietal region with a heavy iron bat. Remained unconscious for several minutes and bled profusely. On regaining consciousness he felt dizzy and weak and had difficulty in talking. He complained of numbness in right hand and foot—could not feel a pin-prick. Four days following injury his skull was opened and one piece of bone removed. Following the operation he had no difficulty in talking and numbness disappeared. Remained well until three months ago, when he struck the left side of his head against a tree while riding horseback. Was not unconscious. Complained of dizziness all day, and on the following day he could articulate with difficulty. The right hand and foot became numb. Could not hold objects in hand.

Previous Diseases.—Had typhoid last summer—three weeks type. Gonorrhea at age of twenty-two and also at present. Chancre at twenty-two. Six months after had nocturnal frontal headaches. No sore throat, no rash. Headaches disappeared after six months of mercury treatment.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix, will you kindly take up the history of this case? This is a very interesting case.

Dr. Mix: The question in this case is whether this man's trouble comes from an old fracture of the skull or whether it comes from an injury sustained many years after the fracture.

The patient is a cowboy from Arizona, and is thirty-four years of age. Twelve years ago he had a fracture of the skull. The left parietal bone was caved in, but the bony fragments were removed and the wound healed without causing any symptoms whatever. He was normal in every way except one; he had a vulnerable point in his skull. In time the unprotected spot was injured.

Two or three months ago, while riding swiftly on his horse, it ran into a tree and he was thrown to the ground. Within two or three hours' time symptoms presented. At the time of the original injury the skull healed in such a manner as to leave a bridge of bone across the large indentation, thus dividing it into a larger anterior area and a smaller posterior one. At the time of the last accident the patient believes this bridge of bone was depressed, and he is certain that it is now more depressed than it was before the accident.

As I said, within two or three hours' time his speech was disturbed, and gradually this disturbance has become more pronounced. The disturbance is a dysarthria, and it is apparently general, since his labials, linguals, and gutturals are equally affected. If anything, his dysarthria is growing worse rather than better.

Besides the dysarthria, he complains of clumsiness and possibly a mild degree of weakness in his right arm. He says he can walk reasonably well.

Physical examination shows normal ankle- and knee-jerks. There is no Babinski reflex on either side. The abdominal and epigastric reflexes are present. The cremasterics are active. The pupils are equal, round, and react to light and accommodation. Strength in the arm and leg on the right side is normal. There is no paralysis or paresis. The tongue is protruded straight, but seems to show a possible atrophy on the right side. Aside from this very doubtful atrophy on palpation, there is no atrophy elsewhere. There are no spasms or convulsions, and no history of either. There are no contractures. His memory is not quite so good as it was before the last accident.

The storm center in this case, then, seems to be over the left hypoglossal center, leading, by irritation of it, to dysarthria. The clumsiness in the right hand might be ascribed to irritation of the superficial cells over the hand center, without disturbance of the deeper-lying motor pyramidal cells. The absence of motor irritative phenomena, such as Jacksonian epilepsy, general epilepsy, and contractures, suggests a very superficial lesion, except at the hypoglossal center. The dysarthria is suggestive of more cortical irritation here than elsewhere.

The diagnosis, as we have made it, is cortical irritation, superficial, over the right hypoglossal center. Whether the pathology is merely a thickened dura, or whether there is some actual compression by the bony bridge, or whether there is a subcortical cyst, remains to be settled when the skull is opened. Against the latter contingency is the grouping of the symptoms.

EXAMINATION OF EYES BY DR. RICHARD J. TIVNEN

(1) *Vision:*

$$\left.\begin{array}{l}\text{O. D.}\\ \text{O. S.}\end{array}\right\}\text{ 20/20ths.}$$

(2) *Position of Eyes:* Normal.

(3) *Excursion:*

$$\left.\begin{array}{l}\text{(}a\text{) Lids.}\\ \text{(}b\text{) Globe.}\end{array}\right\}\text{ Normal.}$$

(4) *Pupils:*
  (*a*) Pupils of both eyes, regular; slight mydriasis of right.
  (*b*) Reactions:

$$\left.\begin{array}{l}\text{(}a\text{) Direct light.}\\ \text{(}b\text{) Consensual.}\\ \text{(}c\text{) Accommodation and convergence.}\end{array}\right\}\begin{array}{l}\text{Normal}\\ \text{both eyes.}\end{array}$$

(5) *Fundi:* Normal.

(6) *Fields of Vision for Form and Color:* Normal.

DR. MURPHY: The history of the injury dates back how far?

INTERN: To the age of twenty-two, twelve years ago.

DR. MURPHY: Has he ever had any symptoms since?

INTERN: No.

DR. MURPHY: This is a peculiar case. I do not know how these head injuries act on the brain, but I do know how other injuries often produce diseases of the joints in tabes. A man will have a lesion in his joint, and go on comparatively comfortable until he has a trauma. After that he will readily develop a Charcot joint. Usually a Charcot joint is associated with the other symptoms of tabes. This man has nothing like that, however. The joints have no relation to his other trouble. It is interesting to see how long these patients can go on without any considerable inconvenience. He has no convulsions, epilepsy, or headaches. The symptoms that usually follow injuries of this kind are: (1) Headaches; (2) attacks of vertigo; (3) epileptic convulsions.

I remember one case that I treated. A baby fell out of the second-story window. The boy was unconscious for three weeks, but had no trouble after that until he was twenty-two years old, when he commenced to have his typical epileptic convulsions. We trephined his skull and relieved his headaches, but did not cure his convulsions. The convulsions are going right on and commencing to become more violent.

We have to interpret the clinical history of this case either as Dr. Mix does, that it is due to the original injury twelve years ago, or that it is due to the last injury sustained two or three months ago. We have in this case some new conditions to deal with. First, he has in the middle of the involved parietal area a depressed linear scar about three inches long, with an almost corresponding absence of the skull. The dura is firmly adherent to the edges of the bone. The dura was evidently lacerated in the original injury, so that, first, we cannot make our flap as conservatively as we would like to, and, second, we cannot take out the bone, because when I come to the central part there is liable to be considerable hemorrhage.

We will make the usual horseshoe-shaped incision, so as to include in it this entire area over the left parietal bone. That flap will measure five inches in diameter. We secure hemostasis in this case by means of deep sutures in the scalp. We are going to remove as little of the skull as we possibly can. I want to make a large osteoplastic flap. The only way good brain surgery can be done is to make a large flap. The trephine is used first to make the opening into the skull, through which we can start to cut out the osteoplastic flap, for which purpose we use the bur. We are not having much difficulty with the bone portion of the work, but we must be very careful in freeing the dura from its adhesions. The dura is very thick. It is a mass of cicatricial tissue. There is no pulsation. See how white it is! One is impressed with that fact at once in taking out the button of bone with the trephine. The button did not come out as quickly as it should. I apparently struck against the adherent dura on the under side, and it retarded the drill. When we were East we saw a number of these bone drills, but I did not think that any of them worked as well as this machine. It was originally worked out by my associate, Dr. Neff.

I am blazing the way for the fracture of the base of this bone-flap, not cutting the soft parts but cutting into the bone enough to try to make it break on this line. The soft parts will act as a hinge. I am trying to get that dura free down there so as to tear it as little as possible. Now I have it freed, and we can break the bone. I did not go through the dura. That is just fine! We break this bone very carefully, because we want to preserve it to fill in as much of the defect as possible.

The dura is not pulsating. It is, as I said, a mass of cicatricial tissue. Whether there is a cyst present or not I do not know. We will not remove any of that dura because we have a defect in the bony portion of the scalp. If we did not have that hole there, we would take out that thickened dura. I will incise the dura, however. There is no cyst connected with the fracture. Here is something! All the resistance ran just across that line—the ascending parietal convolution. I will puncture the brain here with a brain needle. I despise brain needles, yet sometimes I must use one. That did not strike anything resistant—nothing like a cyst. I will change my original plan and take that dura out, because there is enough left to give me a good flap. The brain pulsates now, but the convolutions are flat. The blood-vessels are full. The brain is slightly edematous over the ascending parietal convolution. We will cover this denuded area with gaudaphil, a cellulose tissue.

Let the record show that the dura was enormously thickened and adherent in the position where the bone had previously been fractured, but there were no adhesions between the dura and the pia; that the brain appeared to be edematous; that it did not offer great resistance to touch. A single puncture was made with a small needle; no additional resistance was noted, and no fluid escaped when I removed the stilet. There was no hemorrhage from the dura when it was incised, as it was a white, firm, cicatricial mass. The dura was opened and a piece about three inches in diameter was excised and the defect covered with cellulose paper. We know that excision of the dura gives greater relief from the convulsions than anything else, but whether he will get relief from the type of trouble that he has I do not know. What a decompression will do here remains to be seen. We will replace the bone and tissue flap and suture the soft parts with silkworm-gut and the skin with horsehair.

The tension, remember, is all carried by the silkworm-gut, but an accurate approximation of the scalp is accomplished by the horsehair. All this will be covered again with a bony covering, as nature intended it should be. The wound is dusted with bismuth subiodid and sealed with collodion gauze. The final dressing will be a starch head-piece, because it is light and can easily be removed to make later dressings.

[Following operation there was a transitory mono-paralysis of the right arm and considerable increase in the speech disturbance, lasting for three days. Then improvement began and continued rapidly. Fifteen days after operation the right arm could be used with perfect ease. Speech is considerably better than it was at the time of the operation. We expect an entirely good result.—Ed.]

FRACTURE AND LUXATION OF THE NECK OF THE HUMERUS

HISTORY

Patient, male, aged twenty-one, comes to hospital on account of inability to use left shoulder-joint following a fracture.

August 4th (two months ago) the patient was flying his biplane, when something went wrong with the engine. He shut it off and volplaned to earth. He landed near a barbed-wire fence and ran directly into it. The machine was stopped by the fence, but he and the gas tank were thrown over the fence. He struck a chicken-coop and fell to the ground, landing on his left shoulder. Pieces of the coop and gas tank were on top of him. He was unconscious for fifteen minutes. When he regained consciousness he had an aching pain over the upper third of the humerus. He was unable to move the arm; no pain in shoulder; few scratches about head and back and other minor injuries. He was taken to a hospital, where the trouble was diagnosed as a backward dislocation of the humerus and a double fracture of the scapula. He was anesthetized, but does not know what was done. When he woke up, the shoulder was bandaged so tightly he could scarcely breathe. Over this was a wire-screen cage. The arm was in a sling, with the elbow bent at a right angle, and forearm midway between pronation and supination. Ten days later the dressing was removed and the shoulder looked normal. The doctor then strapped the scapula with adhesive and the arm was held tight to the thorax with adhesive, the elbow and hand being free. Five days later he was examined again, and this time there was a bone protruding just behind the shoulder-joint, which the doctor found difficult to diagnose specifically. Advised massage.

COMMENTS AND OPERATION

DR. MURPHY: What was the diagnosis at that time?

THE INTERN: Backward luxation of the humerus and double fracture of the scapula.

DR. MURPHY: We have taken many skiagrams of this case,—I think six,—and at all sorts of angles. In one skiagram the head of the humerus appears to be out of the glenoid cavity; in another it appears to be in the glenoid cavity; in a third it appears to be partially out. When you look at the patient, it appears to be behind the line of the articulation, which shows the difficulty of interpreting skiagrams. There is no doubt that he has a fracture of the upper end of the humerus which incapacitated his arm. It hangs down by his side, and he can do nothing with it. His muscles are not at all paralyzed, so it is a lesion of the head and neck of the bone. As for the scapula, if anything, one might think, from examining one skiagram, that he had a fracture of the scapula. In another the scapula looks normal and does not deviate at all. It is difficult to figure out the scapula because the distance between the head of the scapula and the acromion is too great. We are unable to get a definite bearing. It looks as though it was fractured obliquely. At any rate, it incapacitated him.

We have two things to do for this man: He must have the ability to lift his arm restored, either by removing the head of the bone and rounding off the upper extremity of the bone, or freeing the head, placing it back in the glenoid cavity, and nailing it to the neck of the bone.

The skiagrams illustrate the various stages of this trouble. One skiagram shows almost the normal position, showing how misleading a skiagram may be unless more than one is made from different angles. The physical examination did not agree with the skiagram. It showed very distinctly that there was a backward luxation of the head of the bone.

We had a case in which there was fracture of the neck (Figs 21, 22, 23, and 24); the head was luxated below the glenoid cavity. We removed the head, freshened its surface, and put it back in

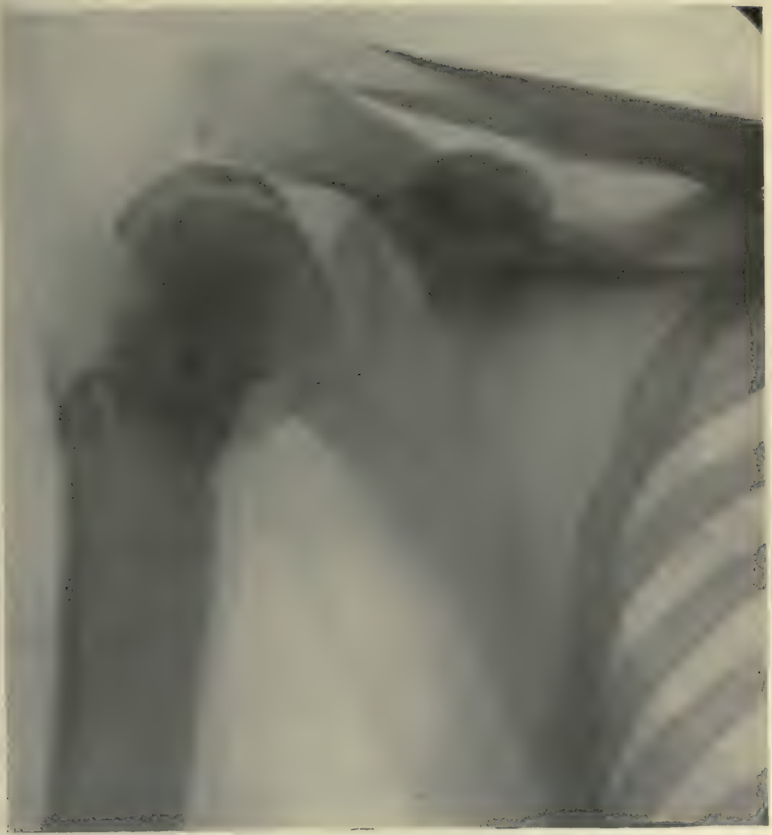


Fig. 21.—Fracture of the upper extremity of the humerus, with backward and inward rotation of head and upward displacement of shaft. The head was also fractured longitudinally, but the fragments had united. In this picture the larger of the two fragments is in front. The upper edge of the smaller fragment is visible above larger one. Head is also subluxated.

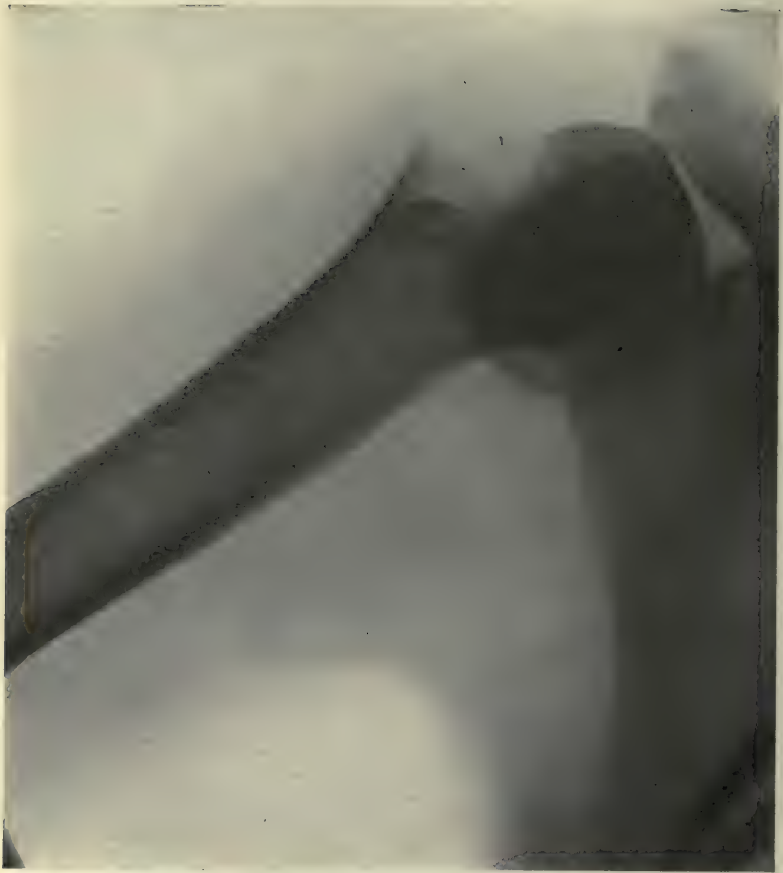


Fig. 22.—In this picture arm is abducted to an acute angle with the body. The head appears to be in the glenoid cavity. Downward and inward rotation is apparent.

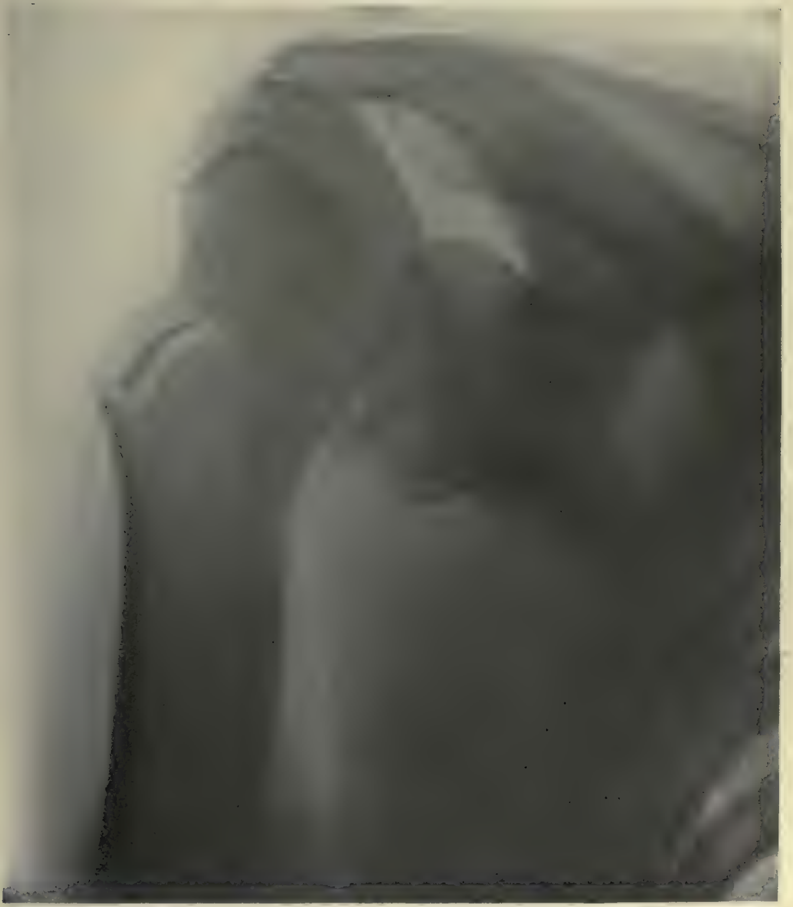


Fig. 23.—Arm is held close to side of the body and slightly forward, so that a portion of upper surface of the shaft fragment is visible. Head is rotated downward and backward so that the two fragments are plainly visible. In this skiagram the head appears to be dislocated upward.

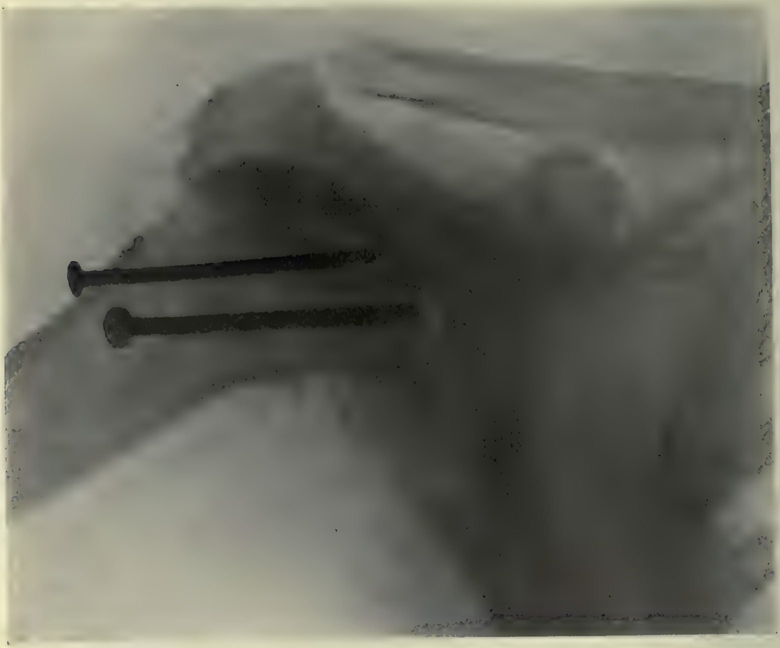


Fig. 24.—The line of fracture is barely visible. The approximation secured by the two wire nails is unusually good. Head of the bone is squarely in glenoid cavity, arm being at right angles to the body. Anatomic result is perfect.

the glenoid cavity, nailing it to the shaft. It filled up with bone and gave us a perfect anatomic and functional result. That is what we may have to do in this case today. The skiagram taken before the operation shows the subluxated head, and one taken after the operation shows the head in its normal position, with the nails in place. Later we removed the nails because they were loosened by absorption of bone around them. The last skiagram shows the final result, a most excellent one.

The head and acromion process in this case appear to be palpable. There also appears to be a considerable callus there at the fractured end of the shaft. That is not going to be an easy operation. How long since this accident?

The Intern: Since August 4th.

Dr. Murphy: Since August 4th, just a little over two months ago, so that he has probably a bony union by this time. The upper portion of the shaft is palpable, and in the position shown by the skiagram. It is in front of the head.

In making the incision here to expose the joint I will make the incision parallel to the posterior border of the deltoid muscle on the posterior surface of the shoulder.

Remember, enormously greater care in asepsis must be exercised in doing bone and joint work than in doing work in the abdomen, or you will have infection. The muscle-fibers will be separated, not cut.

Some of the fibers of the muscle are adherent to the fractured end of the shaft, so that they come in between the ends of the bones. The anterior portion of the head is now in plain view. It looks as though it had been split into two unequal parts and then it reunited. The head is rotated entirely out of position. We will separate these two pieces at the line of fracture, because we cannot possibly get this misshapen humeral head back into its normal position and secure a good functional result. There! it separates of itself. The union was not very firm. It is only eight weeks old. We will use the large piece to make a new head. These deltoids paralyze with great ease and do not restore with any kind of rapidity. When this fracture occurred, it threw the head completely out of the joint. We are in the glenoid space. There is the portion that remained above. We can see where the head belongs—back in here in the glenoid cavity. I can get my finger in, but I cannot replace this head in position without taking it out completely and shaping it. I prefer not to take the head out. Notwithstanding it has repaired you always feel that you know what it does when its vascular supply is retained, even as nature left it. There is the split head.

Now we have things ready. The anterior portion of the head is still in the glenoid cavity. There is the posterior portion of the head. You see it is split. You cannot put these two pieces back. We are going to utilize the half that is in the joint for a new head and dispense with the other half. This will come out entirely. We will nail the retained portion to the shaft. That is our plan of procedure.

Now we want to clean this field out. With a sharp spoon we will take out all this débris of bone that is here, and then I think I will be able to smooth this bone over. As I anticipated when I palpated this bone, a callus has been produced on the end of the shaft fragment, and I must clean that out so that I can rotate the head into position. There is some cicatricial fixation behind here on the capsule. The place in the neck of this bone where this head belongs is easily determined. I have to swing that head clear around to get bone contact. I think I will have to take off a piece of this bone before I can do it. The muscle contraction has been too great to permit us to do otherwise. The head is perfectly free now and we must go back to the original proposition. We will take out the head fragment, freshen the surface, which must contact with the shaft and then replace it in the glenoid cavity. In the case cited above we had to take the head out and replace it in order to secure a normal joint. The glenoid cavity is intact. The fragment of the head which we figured could be used fits well into this cavity. Now we are ready to bring the head and shaft into alinement. The difficulty in restoring this continuity comes from the shortening of the arm muscles. We have replaced the head fragment, and by making strong traction on the arm, we can, by abduction

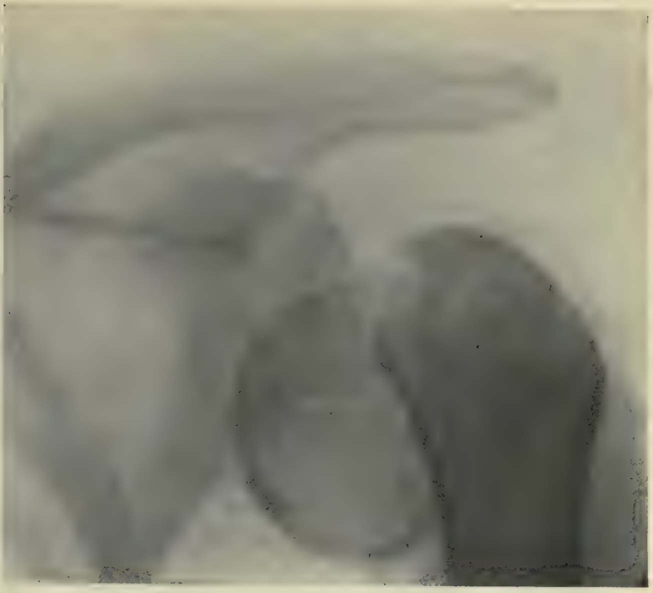


Fig. 25.—Fracture of the anatomic neck of the humerus, with dislocation of the head of the bone into the subglenoid space. The humerus shadows are retouched with pencil, so as to emphasize the relative position of the head and shaft to each other.

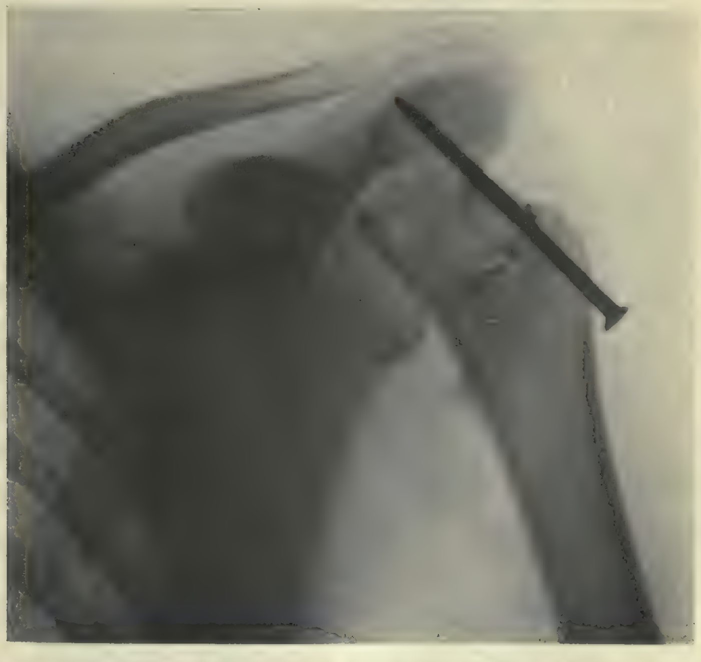


Fig. 26.—Replacement of head of bone into glenoid cavity and fixation of head to shaft by means of a wire nail.

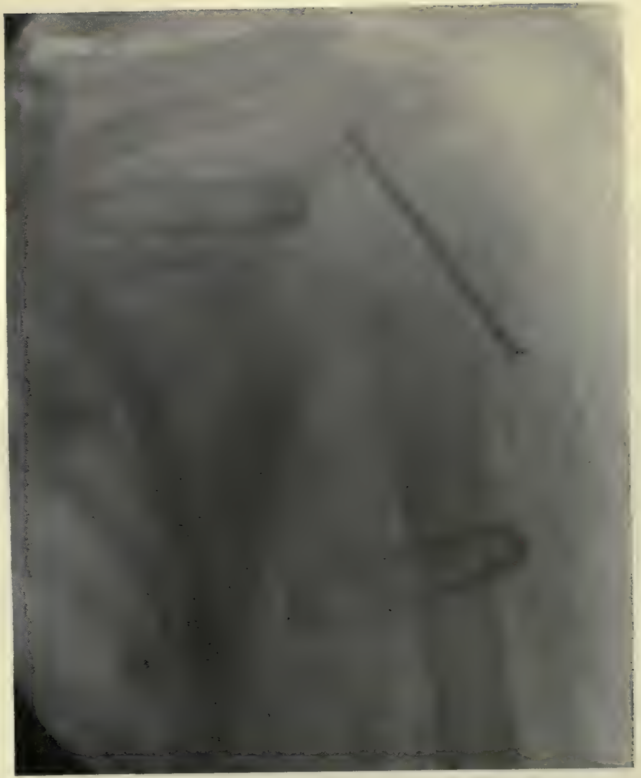


Fig. 27.—Skiagram taken through the plaster-of-Paris cast shortly after the operation. The shadow of the head of the bone is very light and rather indefinite, showing that it was possessed of but little vitality. Comparison with subsequent pictures shows how well the head was regenerated.

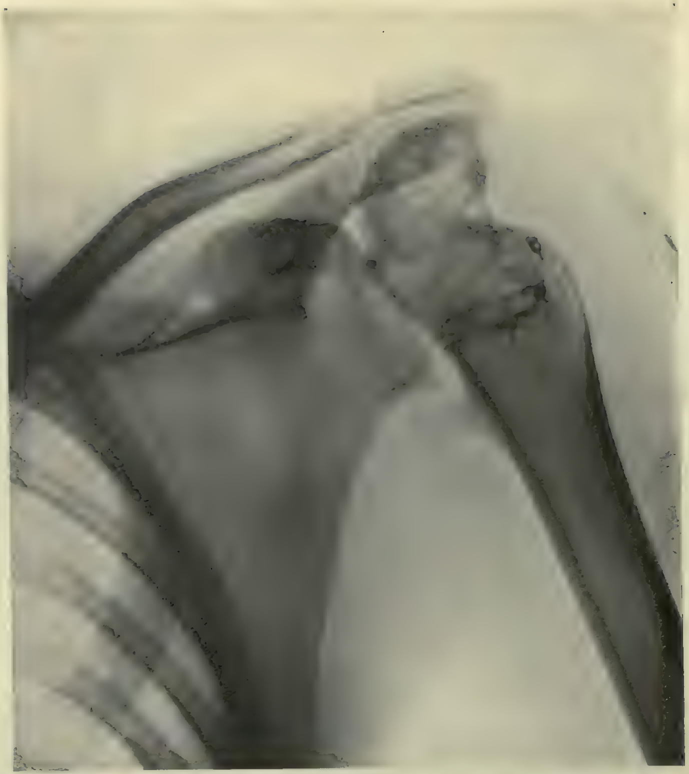


Fig. 28.—Four months later. Nail removed, sutures *in situ*, regeneration of bone complete, with return of function in joint.

and inward rotation, bring the head and the shaft into a normal position. Now you can see that the humerus moves as one piece. "*Es bewegt sich wie ein Stück!*" I secure the approximation with two wire nails driven into the head from the external surface of the shaft. The arm is still abducted almost at right angles to the body, and will be kept in that position for six weeks, so that bony union will be sure to take place. That is all we need to do. We close the wound with deep catgut and superficial horsehair sutures, dust it with bismuth subiodid powder, and seal it with collodion gauze. A large 5 per cent. phenol dressing is applied to take up the secretion and prevent saprophytic decomposition of the fluid that escapes.

VISITING SURGEON: Do you put in a drain, doctor?

DR. MURPHY: If you drain this, you ruin your work. These cases are either sterile or infected. Any place where you have separated bone fragments you cannot drain. If the wound is infected, the operation is futile; the result is *nil*, regardless of whether you drain or not. If it is not infected, there is no occasion to use a drain.

The subiodid of bismuth is merely a desiccating powder. It is not antiseptic.

I am steadying this arm very carefully until all this dressing is done. We will put on a plaster-of-Paris cast, which will encircle both shoulders and both arms, enveloping the whole upper part of the trunk. This arm is kept in extreme abduction, with the forearm at right angles to the arm. The other arm is by his side, and will be kept free below the elbow. He will lie in this crucificial cast, so that the operated shoulder will be immobilized completely.

VISITING SURGEON: How about the tension on the brachial plexus, doctor?

DR. MURPHY: I don't think there will be any tension on that at all. There is no constriction anywhere. We will let him use his hand as much as he desires, but his arm and forearm will be out of commission. Plaster is the surgeon's great friend in this work. We will cut this cast in the usual manner, so that it will not be uncomfortable. Except for the dressing on his shoulder, the cast elsewhere is in contact with the skin.

[The cast was removed four weeks after the operation. The patient had no pain in the shoulder and had joint motion. The cast was replaced. Three weeks later it was removed permanently, and when the patient left the hospital nine weeks after the operation, normal joint motion was being restored rapidly. The skiagram shows a beautiful bony union and regeneration. The nails were not removed.—Ed.]

I want to show you now an arthroplasty we did for an ankylosis of the jaw. [Shows patient.] This boy has had an ankylosis of the jaw since he was seven months old. He is now fifteen. He had an infection in his ear which produced a suppuration in his mandibular articulation, and as a result he had fixation of the jaw. The first time since he was seven months old that he opened his mouth was three weeks ago. We chiseled that ankylosis free, and interposed a flap of the fascia of the temporal muscle to prevent recurrence of the bony union, which was very extensive in this case. We had to remove the coronoid process and a portion of the condyle of the jaw. I had three of these cases in one week, which shows what an opportunity we have for doing something for these patients. [A full description of this case, with illustrations, was given in the December number of the Clinics.—Ed.]

LAMINECTOMY

HISTORY

Patient, male, aged twenty-eight, enters hospital on account of paralysis of both legs and inability to extend the forearm.

July 29, 1911, patient dove into a river after taking a running start of 15 feet. The bank of the river was 3 feet above the surface of the water. The depth of the water at this point was 18 inches, and the bottom was hard and packed with sand. Patient struck on left side of head and shoulder, bending the head sharply to the right. Patient did not lose consciousness and attempted to arise, but could move neither arms nor legs. Experienced no pain. Vesical and rectal incontinence began immediately. On the following Monday patient's temperature was 107° F.; the next day, 103.5° F., and continued this high for one week. Immediately following the accident he noticed complete loss of sensation below the shoulders. Twelve days afterward a large bed-sore was found over the sacral region, measuring 4 by 2 inches. This is almost healed now. Last November (1911) had bed-sores on both heels and right hip.

Four months following injury he was troubled with priapism. Six months ago complained of severe burning sensation from hips down; so severe at times that t kept him awake. One year ago he began to have drenching sweats, more pronounced above the hips than below. These sweats stopped one month ago. Last July involuntary flexion of the legs occurred, immediately followed by violent extension, so severe at times that he would have to be held in bed. The arms would be flexed violently and contract on chest-wall. Lost 50 pounds in weight in five months following accident, but has been gaining in past three months.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix, will you kindly present the neurologic phases of this case?

Dr. Mix: One hot day in July, 1911, the patient went swimming. In attempting to dive, he did not jump out sufficiently far, so that instead of going head first into a hole 10 feet deep, as he intended, he went into a hole only 18 inches deep. He struck on the left side of his head and his left shoulder, and he turned his head sharply to the right. This he distinctly and even vividly remembers, because he did not lose consciousness for a second. Indeed, he said he was not even dazed; but he was instantly paralyzed from the breast-bone down. Moreover, his arms were partially paralyzed. He was catheterized for three weeks after the accident, and he has had bladder and rectal incontinence since. Sensation was lost at once from his shoulders down, involving both arms about equally. In twelve days a very large bed-sore developed over his back, and today, seventeen months later, it is not wholly healed. Other bed-sores developed, one being especially severe over the right hip. The smaller bed-sores have all healed. He had no pain from his bed-sores until three months ago, when he began to feel pain in his back. He has also had a burning sensation in his lower extremities, though he has not a trace of tactile sensation in them.

On physical examination he shows a doubtful Babinski reflex in each great toe, but not a frank Babinski. The knee-jerks are barely present, but traces of a response can be made out in the left quadriceps extensor, and possibly some response in the right. The plantar reflexes were present. The cremasteric, abdominal, and epigastric reflexes were absent. The jaw-jerk was normal. The pupils were equal, of normal outline, and reacted normally to light and in accommodation.

As to motion, there is no voluntary motion in his legs or trunk. He can move his arms with his biceps and triceps, and he can use his shoulder-girdle group of muscles. He cannot, however, flex or extend his fingers, spread them, or bring them together, or oppose his little finger and thumb. At first he could not move his head, but he regained the ability within two weeks. At present the arm and hand muscles which are involved correspond in innervation to the level of the seventh cervical (slightly) and the eighth cervical and first dorsal segments of the cord. Below this level all the voluntary muscles are paralyzed.

There are two levels of *sensory* loss. First, the epicritical level, the level at which touch is felt as touch, but is recognized by the patient as "felt, but not natural," begins in front, at the junction of the second rib with the sternum, and runs around to meet the third dorsal spine. Secondly, the protopathic level, the level at which the sense of touch is entirely lost, starts at the junction of the fifth rib with the sternum, and runs around to join the fifth dorsal spine behind. This line is not bilaterally symmetric, being about an inch higher on the left than on the right side. Below this line all forms of sensation are lost.

Besides these sensory losses on the trunk there are sensory losses on both forearms and hands, these losses very accurately corresponding to the ulnar nerve distribution; in the fingers, for example, involving the little finger and one-half of the ring finger, and following up the ulnar border of the hand half-way up the forearm. Not only does this area correspond to the supply of the ulnar nerve, but it also corresponds to the sensory supply of the eighth cervical and first dorsal segments of the cord.

A further important point is the fact that the cutaneous territory of the second and third dorsal segments is not involved.

As to contractures, both legs are flexed and drawn up. In the beginning the arms were flexed in this position (indicating, with the arms flexed and the hands brought together, the fingers flexed, over the top of the sternum), which is very characteristic. They are still so flexed in part. Both legs are very stiff, but can be passively extended. The arms can be straightened more easily than the legs.

As to atrophies, all the muscles supplied by the eighth cervical and first dorsal segments of the cord are atrophied, more in the left arm than in the right; that is to say, the interossei, lumbricales, small hand muscles, almost the whole extensor group, and the ulnar flexors are especially involved. The legs show merely a general atrophy, not selective, and therefore not indicating anterior horn involvement at a particular level.

In this case, then, there are apparently two entirely distinct lesions simultaneously present—one, a cervical lesion involving the eighth cervical and first dorsal segments of the cord, causing the arm symptoms, and the other a crushing injury of the cord at the fourth dorsal segment, caused presumably by a fracture-dislocation of the third dorsal vertebra, which overlies that segment of the cord. And between these two points more or less normal cord seems to lie. I made a diagnosis of a cervical hematomyelia and compression of the spinal cord at about the level of the third dorsal vertebra.

[Shows the *x*-ray plate.] If you will pass around this plate, you can see a fracture-dislocation at that point [indicating], which is at the level of the third dorsal vertebra. If you will examine the cervical vertebræ, you will note that they are perfectly normal. We must infer, then, a crushing injury at the third dorsal vertebra and another sort of injury at the eighth cervical and first dorsal level. What injury could be acquired here as a result of trauma? Evidently, a hematomyelia. In fact, the sort of injury the patient sustained, with the sharp and sudden bending of his head when he struck bottom, would be eminently likely to produce a hematomyelia. Moreover, the position of the patient's arms is highly characteristic—flexed at the elbow and again at the wrist, and the fingers also flexed, and the hands nearly meeting at the top of the sternum. Shall we find blood in the spinal canal now, seventeen months after the accident? Certainly not. There will be present now only a cyst, largely spreading anteriorly, mostly involving the anterior horn cells—spreading the cord, but not absolutely cutting off conducting power.

Interesting is the fact that for a time the patient could not turn his head. We have merely his statement, but it seems to imply a paralysis of his sternocleidomastoids. These are supplied by the spinal portion of the spinal accessory nerve, which springs from a column of cells in the lateral horns of the cervical cord. Evidently, at first some of these lower-lying cervical cells were involved as a "neighborhood" symptom. In two weeks the head could again be rotated.

We believe, therefore, that this patient is suffering from an injured and compressed cord at the level of the third dorsal vertebra, and from the secondary consequences of an old hematomyelia at the level of the eighth cervical and first dorsal segments.

Dr. Murphy: We will cut down on the spinous processes in the lower cervical and upper dorsal region, and remove them. The incision is made right over the spinous processes. The soft tissues are freed from the vertebræ with a periosteotome.

The spinous processes will have to be entirely removed with bone-cutting forceps, and then we remove the laminæ with rongeur forceps, being very careful not to injure the cord. These operations are very tedious, because we must expose the cord without injuring the dura. Is the fracture at the fourth dorsal vertebra?

Dr. Mix: It is between the third and fourth dorsal vertebræ. The upper level of sensory disturbance comes to the third dorsal vertebra, and the skiagram shows that it is between the third and fourth.

Dr. Murphy: We will remove the laminæ from the fourth dorsal to the seventh cervical vertebra.

Dr. Mix: I want to make a point regarding the sensation. You remember I said that there were two lines of sensation—the epicritical and the protopathic. These were clearly brought out by Dr. Head some years ago, based upon experiments upon his own body. In this man the protopathic line is lower down than usual—it is at the fifth rib in front and the fifth dorsal vertebra behind. The epicritical line of change begins at the sternum in front at the second rib, and runs around the thorax to meet the third dorsal spine. Remember, he feels above the epicritical area, but he does not feel touch as "natural" beneath it, and it therefore marks the level where the trouble lies.

Dr. Murphy: The deviation is at the third dorsal vertebra. I am taking off the spinous process, as you see now, with this heavy bone-cutting forceps going in just deep enough to clear the dura. In doing this operation it is perfectly justifiable to remove the lamina. The point of pressure is at the third dorsal vertebra. There is a hematomyelia above that point, extending well up into the cord. Now, you see, in a comparatively short time I have the cord exposed. There is the position of the anterior compression opposite the third dorsal. You can see where the cord was traumatized. See the absorption that has taken place. The cord is flattened out. I am going to expose the dura all the way. The cord above is distended and does not pulsate at all. We will open the dura in this case to allow the fluid above this point of compression to flow out. See how it gushes out! It is a flood. The pulsation of the cord is rapidly returning. We will have to prevent the fluid from reforming, and we will do that by drawing out through the cord the wall of the hematomyelic cyst and suturing it to the dura with horsehair. The horsehair will act as a seton and allow continued drainage, so that the cyst will not reform. This is the first time we have performed this particular type of operation. We are convinced that the result will be all we anticipate. The dura wound will be closed with very fine catgut sutures.

One runs an additional risk here because we opened the dura. Dr. Mix, I congratulate you on your diagnosis. I put that seton in because I want to get a permanent drainage. I want this fluid to leak permanently out into the subdural space.

Let the record show that there was a compression of the cord at the third dorsal vertebra, that there was a dilatation of the ependyma from the level of the seventh cervical vertebra down to the third dorsal; that the cord was hematomyelic; that there was a gush of fluid as soon as the dura was opened. There was no pulsation in the cord until the ependymal sac was opened. Let the record also show that the findings concurred with Dr. Mix's diagnosis.

This operation was done on a plan worked out some time ago. This was the first opportunity I had of carrying it out.

The muscles and fascia will be reunited with heavy catgut sutures, and the skin with silkworm-gut. We will not put in any horsehair sutures. Bismuth subiodid powder, collodion gauze, and a 5 per cent. moist phenol gauze complete the dressing. No drain; no cast; no support of any kind. These patients are very comfortable. They do not suffer any inconvenience or weakness of the spine from the operation, notwithstanding that the spinous processes and laminæ are entirely removed.

[Stitches removed on the fifteenth day; primary healing. On the sixteenth day there was an inability to move the arms, which was maintained for a period of about a week. Since then he has been very well and can move his arms 25 per cent. better than before operation. One week ago he began to have severe and stinging pains in the legs, of which he is conscious. He feels his bed-sore more acutely. No return of tactile sensation, except that he has a conductivity of the pain sense, probably through the gray matter. Bed-sore on back is almost entirely healed. So far as sense of well-being is concerned, he is feeling fine.—Ed.]

CONGENITAL PYLORIC STENOSIS

HISTORY

Patient, female, aged twenty-two months, enters hospital on account of continuous vomiting and faulty nutrition. Present trouble dates from August, 1911, when vomiting became very frequent. Mother noticed that when child was two days old it vomited and continued to do so at irregular intervals for several weeks. In August the vomiting occurred two or three times a day, usually three hours after eating. At this time the child was being fed every three hours and mother noticed that immediately before time for feeding patient vomited. No signs of pain accompanied the vomiting and no loss of appetite was noticed. Vomitus was dark brown in color, rancid, and offensive. During September the child vomited only three or four times a week. For past two months has been vomiting twice daily and usually before feeding time. Amount given at each feeding is six to seven ounces of milk; amount vomited is greatly in excess. Mother noticed rumbling sounds in the child's abdomen a short time before vomiting. If child misses a vomiting spell, next period the vomitus is greatly increased in quantity and more offensive. Immediately preceding vomiting child is fretful and any excitement seems to bring on the vomiting. Weighs 13½ pounds; weighed 6 pounds at birth.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix, will you please take up the medical phases of the clinical course of this case?

Dr. Mix: The baby is twenty-two months old, the only child of its parents. It was not a blue baby; delivery was normal and without instruments at full term. There is no history of previous miscarriages. It was noticed that from the moment it tried to nurse it was unable to retain its food. It has vomited everything it has eaten from the time of its birth up to the present time, and its mother believes that it never has kept food in its stomach more than a few hours. At first the baby was nursed, then it was bottle-fed. For several months it was treated in a hospital, with some degree of success, but vomiting has never been controlled. Nutrition was so bad at one time that the baby had a very old look, suggestive of the ancient-looking babies with marasmus.

Physical examination was not productive of much in the way of facts. The child is now nearly two years old and yet it weighs only 13½ pounds. Thus it is far from normal in its physical development. The child is quite pale, its lips are slightly blue, and it has an appearance which is suggestive of a mild degree of cachexia. It is not a cross baby; in fact, its good humor is remarkable, when its physical condition is remembered.

Percussion does not show a very large stomach; on the contrary, it is apparently nearly normal in size. The skiagram also indicates that it is fairly normal in development. Possibly the lack of enlargement may be due to the frequent vomiting and its very small feedings. Important, too, and consistent with the absence of demonstrable dilatation, is the fact that no visible gastric peristalsis can be made out. It would seem that the stomach has given up coping with the obstruction at the pylorus. With the stethoscope, too, one misses the squirting gurgle of fluid passing the pylorus. Despite its underdevelopment physically, the child is bright mentally. It is almost uncanny to see a child weighing 13½ pounds saying a few words, trying to talk, and doing baby stunts, such as pointing to its nose, eyes, lips, mouth, and chin when told to do so by its father.

The skiagram shows the vertebræ in the midline. To the left is the shadow cast by the bismuth in the infant's stomach. You will observe that there is no apparent opening toward the right and no apparent development of the pyloric antrum. Undoubtedly there is enough of an opening existing to let some nourishment into the duodenum; otherwise the child would

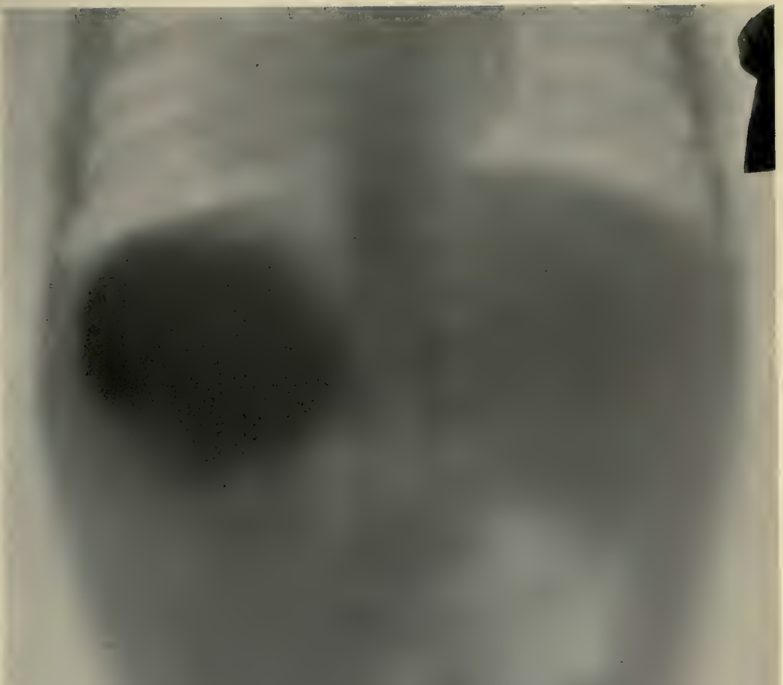


Fig. 29.—The shadow to the left of the spine is cast by the bismuth mixture in the infant's stomach. Mass appears to be isolated, having no connection whatever with any structure to the right. The duodenal cap is absent. There is no evidence of a pyloric opening.

have died. Such a child cannot grow up, for its resistance is *nil.* The parents have been fully advised of the risk of operation, and are willing to take it. Many cases of congenital pyloric stenosis of moderate degree get well without an operation. But this child weighs only 13½ pounds after nearly two years of existence, and is very anemic. What chance will it have if it acquires one of the ordinary infectious diseases of childhood?

If an opening can be made into the stomach, so that the food can pass out if it, the child will probably get well. Gastrojejunostomy will probably be the operation that will be done, unless it turns out when the abdomen is opened that some anomaly which we have not recognized is present. The case seems to be, however, one of congenital pyloric stenosis.

DR. MURPHY: Sometimes the condition continues well into adult life. There are several methods of operation to be considered here—the Heineke-Mikulicz pyloroplasty and the Roux gastro-enterostomy. We prefer to do the gastro-enterostomy in these cases on the Roux plan. It depends on how the patient takes the anesthetic whether we will do it. It also depends on what we find.

Doing the operation on the Heineke-Mikulicz plan has not been very satisfactory. It consists in making a transverse incision through the stricture on the anterior wall of the pylorus and then, by suture, convert it into a longitudinal wound. That gives the best result provided the stomach is fairly well dilated. Finney's pyloroplasty practically makes a new pyloric opening, utilizing both the duodenum and the stomach. The incision is made in the wall of the duodenum on the stomach side, and a similar incision in the stomach-wall, each about three inches in length, beginning the incision in the stenosed pylorus. The edges of these incisions are united, as is customary in all stomach and bowel operations. If necessary we could do a gastro-enterostomy later just as easily as we do it in the adult. The objection to this operation is that regurgitation often takes place back into the stomach. The best thing to do is to divide the jejunum four or five inches from its beginning, anastomose the distal end with the stomach-wall, and suture the proximal end into the side of the jejunum, a few inches from the stomach opening. That is a nice operation if the patient can stand it.

I do not like to operate on little children. This is one of the cases in which I would like to use chloroform, but chloroform is difficult to give, and the mortality from it is high. I recall the case of a little boy on whom I was to operate in my office. He had been told not to eat anything before coming to the office. He was not given any breakfast, but just before he came downtown he went into the kitchen and the cook gave him a big meal of meat. As soon as we began to give him the chloroform he began to get cyanotic. My assistant, the late Dr. Lee, raised him up by the heels and shook him, and out came all the food he had eaten. Only for that he probably would have died and the death would have been charged to the chloroform. It was a case in which we could not have held a postmortem and the father had enough political influence to hold us guilty on the charge of chloroform asphyxiation.

I never got familiar with operating on children, because it has a bad effect on my nervous system. I have seen the late Dr. Gunn, in the pre-anesthesia period of surgery, tie the children in a chair and operate on their palates without an anesthetic just because the child was not big enough to strike him. It just makes me creep now to think about it.

We approach the stomach through the usual incision, going through the right rectus muscle. That gives us a good exposure of the pylorus.

There is the pylorus. It is a firm, hard band. See the ring! There is the ligament of Treitz. When I see that I know just where I am. The transverse colon and omentum are turned up over the epigastrium, and an opening made in the mesocolon at a bloodless area within the loop of the middle colic artery. A fold of the posterior wall of the stomach is drawn through this opening and fastened to the cut edges of the mesocolon with a few tacking catgut sutures, to prevent hernia and constriction of the stomach by cicatricial contraction. A gastro-enterostomy clamp is placed on this fold of stomach and another one catches a loop of jejunum close to its origin. The clamps are laid side by side and surrounded by gauze pads. With a continuous Lembert suture of Pagenstecher linen the stomach is sutured to the intestine for a distance of about an inch and a half in this case.

Both stomach and intestine are now incised down through the mucous membrane and the edges of the incisions are united all the way round with catgut, the suture passing through all the coats of the stomach and bowel; a Czerny stitch, to give a firm apposition and to stop bleeding.

The clamps are now removed, and the Lembert suture is completed around both bowel and stomach. That makes a firm anastomosis and insures against leakage.

That completes the operation.

Shall we do an entero-enterostomy here? No. I feel that this child has gone through this operation very nicely and any time later we wish we can do an entero-enterostomy. The abdomen will be closed in the usual manner.

Let the record show that there was a very highly developed pyloric stenosis, and that a posterior gastro-enterostomy was done in the usual way; that we left about 5 inches of a jejunal loop for the purpose of some time later doing an entero-enterostomy, if necessary. We can do it without difficulty any time in the life of the child. I think that any improvement that will come to this child as a result of this operation will be almost immediate and great.

Let the record further show that Sister Victorine reports the sponges counted and correct.

There was no blood in the vomitus—just a few shreds. That is where the danger is, in hemorrhage from the stomach. That is why I put in an extra row of stitches.

[Almost immediately after operation the baby retained most of its food, consisting of chicken broth and albumin and later peptonized milk. On the sixth day the baby developed a pneumonia on the right side. Temperature rose to 105.2° F., respirations 50, pulse 160. On the fifth day of the attack the child began to improve. On the twelfth day after the operation its temperature is 99.6° F., pulse 128, respirations 42; child is able to cough rather vigorously, resists being handled, and shows considerable spunk. For the last five days has been given 3 drams of peptonized milk every half-hour during waking hours, with no vomiting. Food is now being increased to 4 drams every half-hour during waking hours. At first salines were given, but were later discontinued. The absence of vomiting shows clearly that the food is passing through the opening in the stomach into the intestine. The child's stools are normal in amount and yellow in color. She is doing splendidly now.—Ed.]

LAMINECTOMY TWO YEARS AFTER INJURY

HISTORY

Male, aged twenty-four, plasterer, comes to the hospital because of paralysis of both legs and loss of control of vesical and rectal sphincters. Tuesday, October 10, 1910, while at work, he fell a distance of about 18 feet, and while falling, his back struck on a projecting body. He landed on a cement floor, but does not know in what position he was when he struck the floor. When picked up he was unconscious. When he regained consciousness, after a few moments, he had a severe pain in his back, in the lower lumbar region. This pain has been present ever since the accident, and is worse at some times than at others. The patient cannot move his lower limbs, and he has no sensation in them. Since the accident he has not been able to urinate voluntarily, and he has had involuntary bowel movements.

Physical Examination.—Complete paralysis below the level of the first lumbar vertebra. No zone of hyperesthesia above the level of the lesion. Loss of sphincteric control of the bowels, with inability to urinate. Complete abolition of all the reflexes of the lower extremity and also of the cremasteric. Faradic response fair in right lower extremity, feeble in left, showing that the axones are alive.

COMMENTS AND OPERATION

Dr. Murphy: I will ask Dr. Mix to take up the analysis of the case from the neurologist's standpoint.

Dr. Mix: We must first examine the reflexes. As to the *tendon-reflexes:* the left patellar is absent; the right is reduced, but is present; the Achilles jerks are both absent. Thus, then, on the left side of the body the tendon-reflexes are absent, whereas on the right side they are present, but excessively reduced. As to the *superficial reflexes:* both the epigastric and abdominal are bilaterally present; both plantar reflexes are present, but the left is better than the right. As to *pathologic reflexes:* the Babinski toe-phenomenon is present on the left side, but not on the right; the left-sided Babinski is very peculiar in this respect: pressure in the groin, at the origin of the gracilis and of the adductors longus and brevis, elicits the Babinski reflex. Though extension of the great toe can be called for in many ways, I have never known of a case personally, or in medical literature, where compression of the origins of the adductor muscles against the pubis called for the toe-phenomenon.

The only vasomotor phenomenon of interest is a very pronounced dermatographia, equally intense on both sides of the trunk.

Sensory Disturbances.—The muscle- and joint-sense is completely lost in both legs, for all muscles. The tactile sense is entirely absent, yet touch can be felt as an increase in the burning which is subjectively present in the legs. In other words, when he is touched, he becomes aware of it by reason of his perception of an increase in the burning which is always present. This would indicate that he has some conductivity of sensory impressions, and I strongly suspect that these burning sensations are conducted upward through the gray matter of the cord.

In cases of transverse myelitis and transverse injuries of the cord the limitations of the sense of touch mark the level of the lesion. In looking for these limitations attention must be paid to epicritical and protopathic sensibilities. Protopathic touch is primary tactile sensation—a mere perception of touch. Epicritical sensibility to touch is something more than mere primary tactile sensation: it is an apperception rather than a perception. Memory and judgment enter into its composition. Now, in transverse lesions the epicritical faculty is lost at a higher level than the protopathic; and the epicritical loss of tactile sensation usually marks the level of the lesion very accurately. In testing protopathic sensation you say to your patient: "Do you feel?" In testing epicritical sensation you say: "I know you feel this, but does it feel *natural?*"

In this particular case, on testing sensation we find tactile sensation beginning to seem "unnatural" along the supply of the ilio-inguinal and iliohypogastric nerves. These nerves spring from the first lumbar segment of the cord, which segment lies opposite the twelfth dorsal vertebra. Therefore the lesion lies at this level.

Testing further, we find protopathic sensibility present on the thigh beneath the groin, in the genitocrural territory, but when we get into the anterior crural territory, all tactile sensation is lost. If we depended on an absolute tactile anesthesia for our localization, we should place the lesion opposite the first lumbar vertebra, and we should err.

As to *motor* losses: We find a very slight degree of motion in the right leg from the hip, mostly in the quadriceps extensor, but on examination we find it is an involuntary associated movement. When he tries to lift the leg, this slight motion takes place. He also has involuntary extension movements in the left big toe, this being merely an involuntary, self-excited, Babinski toe-sign. He has told us that when his penis is erect he can move it, but we have not tested this statement. In general, aside from some involuntary movements and pathologic reflexes, all motion is lost below the first lumbar segment of the cord.

Some *atrophy* is apparent in the right leg, for it is surely smaller than the left. We find that the loss is in the anterior tibial group and in the quadriceps extensor. In general, however, there is little or no atrophy and little or no selective atrophy; neither is there any fibrillary twitching.

Contractures are not present. There is some stiffness in the legs, but only a very little.

As to *faradism:* I got responses in the muscles of his right lower extremity, but in the left leg the responses were feeble, slow, and very deliberate. The fact of response at all is of vital importance.

The conclusion is that the patient has a crushing injury of the cord, affecting the first lumbar segment, caused by a fracture-dislocation of the twelfth dorsal vertebra. It may seem like splitting hairs, but the line of force seems to run from the left posterior to the right anterior portion of the cord, indicating that the squeeze came from the left front of the body, pressing backward and to the right, or that posteriorly the right lamina is pressing forward and to the left. The predominating lesion is on the left side of the cord, because: (1) The Babinski toe-sign is on the left side; (2) the knee-jerk is lost on the left side; (3) the faradic response is less on the left side; (4) the involuntary motion from the irritative lesion is mostly on the left side. It is likely that an operation will give some improvement, but the amount of improvement cannot be foretold. The cord is not severed.

Dr. Murphy: That gives you the presentation of the case from the neurologist's standpoint. In the presentation of the case from the surgical standpoint, what we want to know is whether or not the cord is severed, and whether or not the removal of the pressure on the cord will give a restoration of function. We know that in a transverse pressure myelitis, if the nerves or the spinal cord are compressed, the result is an inflammatory reaction—edema, swelling, infiltration, and suspension of transmission. I recall a case I had in 1886. The man fell from a scaffold some 12 feet high, landing on his buttocks. He worked that day and he worked the next day. In thirty-three days he had a complete paraplegia—a paralysis of the legs and a paralysis of the sphincter of the bladder. He had no deformity of the spine, no evidence that the spine had been compressed, but he had no faradic reaction and complete suspension of transmission of motor impulses. He was not operated on,—just treated with rest and tonics,—and in fourteen months he was back at his work with a complete restoration of function in all the muscles. I cite this as an evidence of complete restoration of motor function after suspension of transmission.

In regard to injuries of the spine, cases have been reported by Harte, of Philadelphia, and Fowler, of Boston, of division of the spinal cord with a complete restoration of function. However, when the histories of these cases were analyzed and the symptoms were analyzed, it was evident that they were not cases of complete division of the cord—only partial division of the cord (in Harte's case). A severe injury to the spinal cord does not always produce a complete division of the cord, as reported by Fowler. His case shows exactly the same variance in the examination and the analysis of the symptoms as does Harte's.

In the true cord there are none of the conditions present for regeneration, because the axones are without a neurilemma. When the axone is cut in the true spinal cord, it never regenerates. Of recent years we have had two distinct classes of results in the treatment of divisions of peripheral nerves. If we unite the nerves without the intervention of connective tissue and with axonal contacting and got complete regeneration; if we unite the nerves with their bulb ends retained, we do not get a regeneration, because these ends are covered with connective tissue, which prevents axonal contacting. The men who unite the nerves without the interposition of connective tissue between the cut ends of the nerves had regeneration of the axones and complete restoration of function, and the men who suture the nerves with the connective tissue interposed secured no more return of function than if they had sutured two pieces of cheese together, because the axone could not pass through the interposed connective tissue. The nerves of special sense have no neurilemma and are therefore incapable of regenerating themselves.

Now, then, that is all you really have to keep in mind from a surgical standpoint. The factor in injuries of the spinal cord which is of such importance in making a differential diagnosis is whether the nerves are divided or whether there is only suspension of transmission. It is our duty to relieve these nerves of the pressure on them. Many times the patient has an immediate and complete and annular paralysis of motion and sensation on the same level for the entire circumference of the body.

Regarding the technic of the operation, the first question is, Can we remove the spinous processes and with these the laminæ? and the next question is, Can we remove the spinous process and leave the laminæ? If we remove these structures, does it impair the strength of the spinal column in any way? It is a well-known fact that one can remove the laminæ and spinous processes without doing injury to the column. It simplifies the operative procedure to remove the laminæ and the spinous processes, and one can remove them readily in the majority of cases and without detriment to the patient or traumatizing the cord.

This man has some lateral luxation of his spine at the twelfth dorsal vertebra. We will remove the spinous processes of the tenth, eleventh, and twelfth dorsal and the first and second lumbar vertebræ.

We will make a longitudinal incision directly over the spinous processes of the last three dorsal and upper two lumbar vertebræ, the patient lying partly on his left side and partly on his chest, and the right leg being drawn up in extreme flexion so as to stiffen his body and to throw his spinal column backward, thus making it more accessible for our work. The incision extends down to the vertebræ. The soft tissues on either side of the spinal column are then retracted, so that the spinous processes can be removed with a heavy bone-cutting forceps. We will remove the spinous processes of the tenth, eleventh, and twelfth dorsal vertebræ down to the level of the laminæ. We find that it is the eleventh dorsal vertebra that is particularly involved, and that it is here that the spine is angulated, the tip of the angle being directly backward. The eleventh dorsal vertebra is also luxated backward. With the rongeur forceps we will very carefully remove the laminæ of the tenth, eleventh, and twelfth dorsal vertebræ, so that the spinal cord will be exposed freely. There is not much bleeding, most of it coming from the bone. This we can very easily check by putting in a little Horsley wax, pressing it firmly against the bone. This wax is also useful to cover over the sharp edges which might cut the dura. The backward curve in the spine is not very evident, its highest point being opposite the eleventh dorsal vertebra. The dura is very much thickened by the development of fibrous tissue, and we shall endeavor to remove some of the ragged edges with the scissors. Of course, we do not want to penetrate the dura, because if we did, we would have the additional danger of infection of the spinal canal.

The cord is not pulsating above and below the luxated vertebra, and as we remove more laminæ we find that the tension of the cord gradually is disappearing and the pulsation is again becoming evident in this area. There was, therefore, in this case not a complete division of the cord—merely a compression which we have to relieve in this manner if the patient is to regain any use of his paralyzed extremities.

We have now relieved the cord of this pressure, excised the redundant connective tissue, and the next step is to close the wound.

It is not necessary to cover over the spinal cord here with any foreign bony material, because of the heavy pad of muscles normally present, which is sufficient protection against injury, and during the process of repair the patient wears a leather jacket to prevent flexion of the column. We will suture the muscles with silkworm-gut, suturing them well over the spinal cord. The sutures will also include the skin. Bismuth subiodid and collodion gauze seal the wound. Over this we will place a very large dressing, which will be held in place by a roller bandage encircling the patient's body. There is no further dressing needed to protect the patient's back.

We perform all our laminectomies in this manner, and our results have always been very satisfactory, both to the patient and to ourselves.

VISITING DOCTOR: What is the prognosis?

DR. MURPHY: The prognosis is that he will have a very material improvement.

VISITING DOCTOR: If the operation had been performed immediately after the injury, what would have been the result?

DR. MURPHY: The patient would have been in his grave long ago. He had trophic sores. When these are present, the cases do not do well under immediate operation. His trophic conditions are splendid now. His sores are all healed up.

VISITING DOCTOR: Many surgeons consider it advisable to open the bladder for vesical drainage after such an injury—what do you think of that?

DR. MURPHY: That is the next best thing to not doing anything. It does not do any good. I never open the bladder. We put the patient to bed, then we pad his popliteal space, pad his spine under the small of the back, and that takes the pressure off the high points and prevents a sacral skin necrosis. The first place these patients get a necrosis is over the sacral region, and the next place is over the heels. We put pillows under the heels and prevent a necrosis there. These patients more commonly die from infection of the bladder and ascending infection into the kidneys—they rarely die from the fracture of the spine. We avoid necrosis by an equalization of the pressure and by frequent changes of position. If you will wait until the bladder fills, you will find that it will empty itself of its own accord. You will be surprised how little trouble there is in the first few days, if you just wait until the bladder has the urine under high tension. You can stimulate the voiding of the urine by gently massaging the prostate. It is only a question of time in the management of these cases. If you open the bladder, you are very liable to get an infection from the outside from which the patient will die. *We never catheterize these patients.*

We will keep this man on his side for the next few hours and then we will turn him over on his back.

Let the record show that there was a fracture of the lamina of the first lumbar vertebra, with an inward luxation of the edge of the lamina and a pressure on the cord at this point.

Let the record further show that the spinous processes and laminæ of the last three dorsal vertebræ were removed and that the mass of connective tissue which rested over the dura was carefully dissected out; that the cord was freed; that the dura was not opened, so that there is practically no danger whatever of an infection of the spinal cord. [Primary union. Left hospital in four weeks. No material change in nervous symptoms. Has not been heard from since.—Ed.]

HOUR-GLASS STOMACH

Dr. Murphy: Dr. Mix has a very interesting case about which he wishes to talk to you this morning and show you some fine *x*-ray plates.

Dr. Mix: This man, fifty-nine years of age, referred to me for diagnosis by Dr. Pelton, of Elgin, Ill., has had pain for twelve years in the left gastric region. He complains of pain and nothing more. He says he has "lived with it" twelve years, but hates to contemplate passing the remainder of his days in its company. The pain came on two or three hours after eating. It was relieved when the stomach was full, and relieved most after a very hearty meal, but the taking of small quantities of food caused no relief of the pain. Were the case a duodenal ulcer, we might have a history of pain two or three hours after meals, but the pain would ordinarily be relieved by a little food, and it would be a diffuse pain, to the right of the midline, in that area a little to the right of and above the navel, which some day we are going to call the *duodenal area*. The pain, however, was *left-sided*—in the gastric area.

Such a pain might be referable to the pylorus, but if it were an ulcus, we should expect something more of a history than twelve years of daily pain. A pyloric spasm due to any visceral cause could scarcely have so persistent a course.

Physical examination was negative. The stomach was not specially large, and was in a location within the normal range of variation. Visible gastric peristalsis could not be demonstrated. On passing a tube several hours after eating I abstracted some stomach-contents, showing that there was food retention. Apparently the stomach-tube went in the normal distance. A test-meal showed absent hydrochloric acid and the presence of lactic acid, but there were no Oppler-Boas bacilli to be found, and blood was not present, either microscopically or by the chemical test, neither was occult blood in the feces. A motor meal showed a small residuum after five hours.

The conclusion seemed inevitable that there was food-block somewhere. Evidently the duodenum was not at fault symptomatically or by physical examination. The pylorus was apparently patent, and certainly was not sore. The food-block and the pain under the left border of the ribs suggested to me a possible block in the stomach itself, and hence the possibility of an hour-glass stomach occurred to me. In the old days, for our diagnosis of this condition we were dependent upon the abstraction of stomach-contents from two pouches with two differing acidities, or of outlining with dilatation of the stomach two bulging pouches with a constricted portion between. Now all we need to do is to make careful *x*-ray pictures, with our minds on the pathologic condition we are after. Hence I had three pictures taken, and here they are.

[Shows *x*-ray plates of stomach.]

The first picture (Fig. 30) was made five minutes after the ingestion of 16 ounces of buttermilk containing 2 ounces of bismuth subcarbonate. It definitely shows a shadow across the gastric area. [If this plate is contrasted with the *x*-ray plates elsewhere in this volume showing the full stomach, the shadow will be immediately apparent.—Ed.] The second plate (Fig. 31), made fifteen minutes after the first, shows the food passing from the first half of the stomach into the second half. The third picture (Fig. 32) shows the condition two hours after the bismuth was given. It is very clearly a constricted stomach—an hour-glass stomach.

In this case the correct diagnosis before the plates were made was had in mind, because it was appreciated that there was food-blockade with gastric peristaltic cramp not due to duodenal block or ulcer, and not due to pyloric spasm or blockade; the *x*-ray pictures were then ordered, with some confidence as to what they might disclose.

What caused this form? Probably a round ulcer some twelve years ago. There is no evidence of malignancy; no special

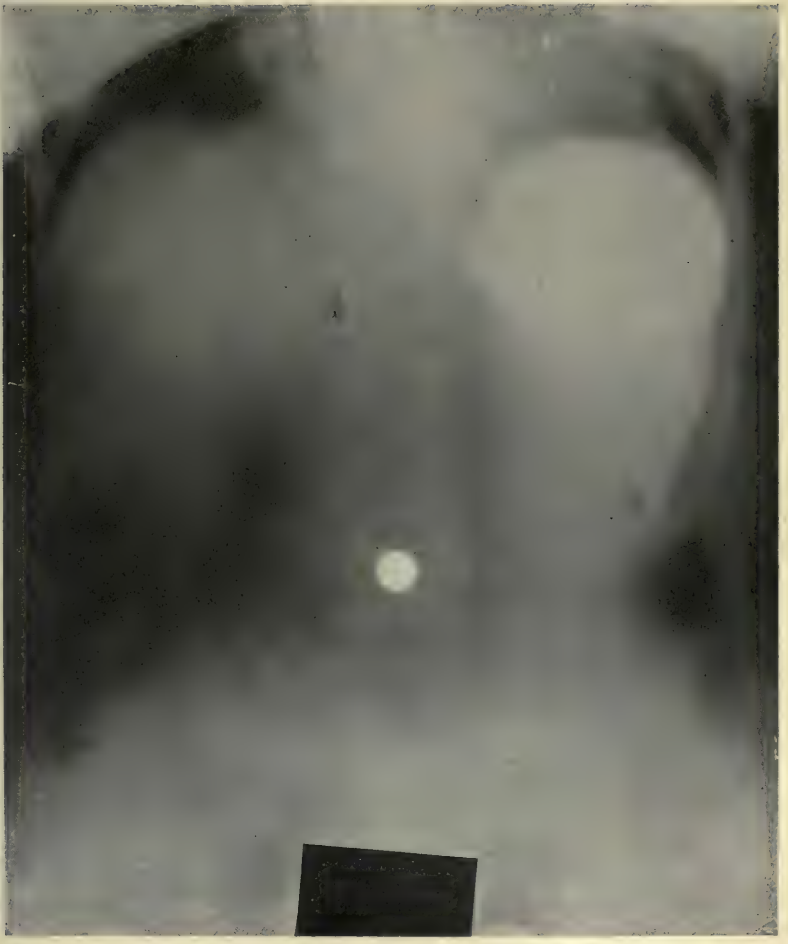


Fig. 30.—Skiagram made five minutes after the ingestion of 16 ounces of buttermilk containing 2 ounces of bismuth subcarbonate. It definitely shows a shadow across the gastric area.

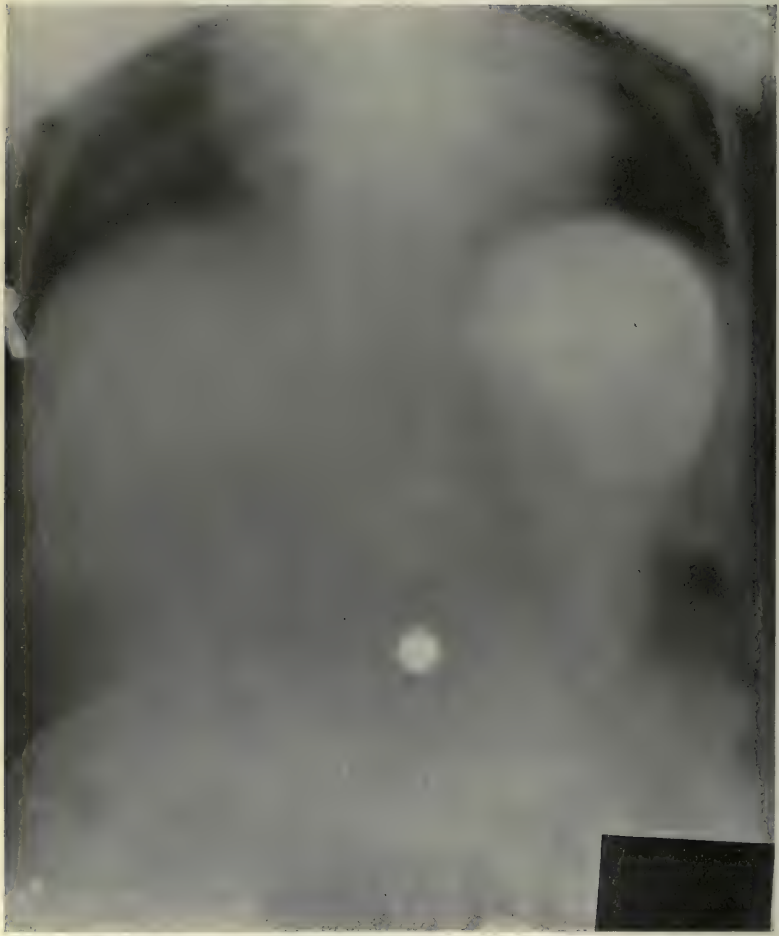


Fig. 31.—Skiagram made fifteen minutes after Fig. 30, showing the food passing from the first half of the stomach into the second half.

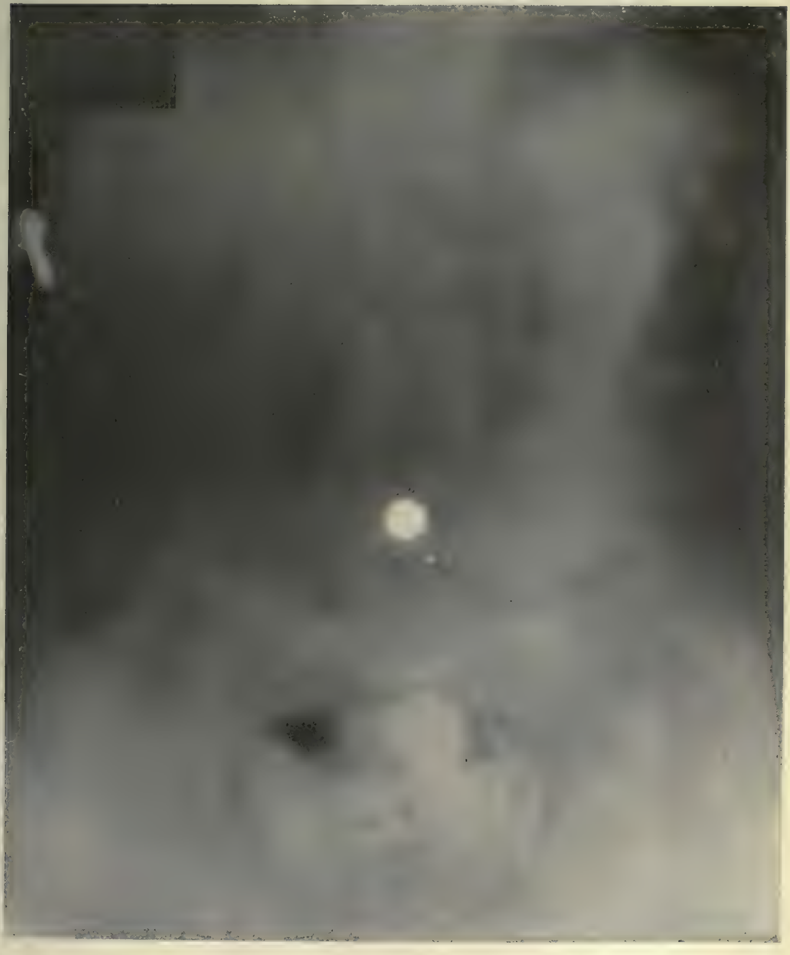


Fig. 32.—Skiagram showing the condition two hours after the bismuth was given. It shows clearly the constricted stomach—an hour-glass stomach.

emaciation, though the patient is rather spare in build and has been clearly under weight for years; no cachexia; no special anemia, his blood examination showing 80 per cent. hemoglobin and about 4,500,000 red blood-corpuscles per c.mm. Carcinoma and ulcer are the usual causes of the acquired form of hour-glass constriction. No proper history of ulcus exists in this case, it is true, but exact information as to the details of the symptoms of onset is necessarily lacking, the memory of daily pain for twelve years blotting out the memory of the pain at its onset.

SURGICAL CLINICS
OF
Volume II JOHN B. MURPHY Number 2

ESSENTIAL HEMORRHAGE OF THE UTERUS—HYSTERECTOMY

DESCRIPTION OF DR. MURPHY'S OPERATION FOR HYSTERECTOMY

HISTORY

PATIENT, a female, aged thirty-six, comes to hospital on account of uterine hemorrhages.

Menstruation began at fifteen years, was very regular, every twenty-eight days, flowed four days, and a moderate amount. Her first baby was born seventeen years ago, when she was nineteen years old. Labor normal. Since that time the flow has been a little more profuse than before. Eight years later (1904) her second and last baby was born. She received a slight perineal laceration at this time, which was not repaired. Ever since then she has been irregular, her menstrual periods coming on every twenty-one to thirty-one days. The following spring (1904) she had her first uterine hemorrhage. The period was a little delayed, but began as a normal menstruation. Instead of flowing four days she flowed seven days, and the quantity each day was double that of any day in her normal period. She was very much weakened by the loss of blood. Six weeks later she contracted measles, and following this had an acute nephritis. She was ill about seven weeks, during which time she had albumin and casts in the urine and her blood-count showed marked diminution in the number of red blood-corpuscles and in the hemoglobin percentage. Her doctor said it was a secondary anemia. Some time between the spring of 1904 and the fall of 1911 she had one uterine hemorrhage. Does not remember the exact date. The duration, loss of blood, etc., were the same as at first. (Her general condition in those seven years was good.) In November, 1911, she had her third hemorrhage, a fourth in February, 1912, and the fifth and last in June, 1912, five months ago. The two hemorrhages in November and February lasted about a week, but the one in June lasted fifteen days. During the first week the flow was very profuse, both day and night. During the second week the flow was profuse during the day only. At night, while she was lying down, the hemorrhage ceased. She was very much weakened by this loss of blood, and she has not felt quite as strong since.

Four weeks ago she had an illness lasting ten days. She had an evening temperature of 103.5° F. No sore throat. No digestive or abdominal symptoms. No chills or perspiration. She was treated for malaria, with large doses of quinin. Two weeks later the same condition recurred and lasted twenty-four hours, with a temperature of 102.5° F. Blood examination for plasmodia was negative.

Her appetite is good. Bowels regular. She has lost ten or fifteen pounds in weight in the last five months. No cough; no night-sweats. No history of tuberculosis or malignancy in the family. No history of bleeding (hemophilia) in the family.

COMMENTS AND OPERATION

Dr. Murphy: When did she have the first uterine hemorrhage?

Intern: In 1904, following the birth of a baby.

Dr. Murphy: She has had her menstruations regularly, is that right?

Intern: Yes.

Dr. Murphy: I want to see how these hemorrhages fit in with her menstruations. She had her last baby eight years ago?

Intern: Yes, doctor, in 1904.

Dr. Murphy: After this confinement she had the first uterine hemorrhage?

Intern: Yes.

Dr. Murphy: How long after?

Intern: She does not know. The child was born early in 1904, and she had the hemorrhage in the spring of 1904.

Dr. Murphy: What I want to bring out about the hemorrhages is this—the menstruation is regular, she did not have an intermenstrual flow, she had the hemorrhages at intervals, and she has had five in eight years. That is not a typical history of either a carcinoma or a fibroid. If she has a fibroid, she should have had an increase in the menstrual flow uniformly, unless it were a serous fibroid: then she would not have an increased flow. That is not an alarmingly large number of hemorrhages—only five in eight years. But the hemorrhages are occurring more frequently —one in seven years, and four in seven months. Subserous fibroids do not give the typical fibroid menstrual history. In the typical fibroid the menstrual periods occur regularly, but are longer in duration and the flow is increased in quantity, but they never have an intermenstrual flow—they never flow between the periods. This patient has never flowed between the periods. The uterus is freely movable in the pelvis. It is not enlarged. Now the question is, has she retained a portion of the placenta from her last birth, eight years ago? Has she a submucous fibroid? Has she a growth in the uterine wall from the retention of that placenta, or has she a deciduoma? Has she a carcinoma or has she an essential uterine hemorrhage? I think it is an essential uterine hemorrhage. I have seen four of these cases of essential uterine hemorrhage—in two a hysterectomy was performed, one case was simply packed, and in the fourth we had an opportunity to do a postmortem. The uterus was not materially enlarged; the mucous membrane was perfectly smooth; the muscularis was firm; the peritoneum was normal. Four weeks before this patient died her doctor tamponed the uterus to stop the hemorrhage.

When I examined the patient a few days ago the uterus appeared to be of normal size, but slightly more anterior than usual. The tubes and ovaries were apparently normal. The cervix was not eroded or thickened.

We will do a hysterectomy in this case, leaving both the tubes and ovaries.

The method of abdominal hysterectomy which we employ is original with us, so far as we know. We have been performing this operation for the past ten years and published a description of the operation in the Illinois State Medical Journal at the time. It possesses so many points of advantage over the commonly accepted methods that we have become convinced of its superiority.

The generally adopted line of procedure, by attack from the anterior side of the uterus, involves a number of difficulties and possible dangers which we believe are obviated by the newer posterior method. The most serious of these is that of injury to the ureters, which lie close to the sides of the corporocervical junction and anterior to it. Because of their proximity to the uterus, one of the ureters may be included in the mass ligature, when the latter is passed around the uterine artery from in front, or it may be divided by the anterior incision through the corporocervical junction. That these accidents are possible is proved by the fact that certain advocates of the anterior operation place catheters in the ureters as a preliminary procedure and they are recorded in the literature.

Another danger in the anterior method is the occurrence of secondary hemorrhage, caused by the slipping or loosening of a mass ligature, which includes not only the uterine or ovarian arteries, but a considerable quantity of the surrounding connective tissue. This is more likely to happen when catgut is used than with unabsorbable materials, as the catgut is quickly attacked by the tissues and becomes soft and slippery within a short time after its insertion; even chromicized catgut may slip and give way before coagulation has taken place in the vessels.

The ideal method of controlling hemorrhage from the uterine and ovarian arteries, therefore, and the one which we employ in this operation, is the ligation of the isolated vessels, as one would the radial artery in amputating the forearm. This renders secondary hemorrhage practically an impossibility.

The technic of the posterior operation in detail is as follows:

1. Aseptic preparation of the abdomen and vagina.

2. Trendelenburg position.

3. Abdominal Incision.—A vertical incision, 5 to 8 inches long, is made through the inner border of the sheath of the left rectus muscle. The fibers of the muscle are displaced outward with the handle of the scalpel and the peritoneum divided on the slant between the two tissue forceps which hold it. The incision

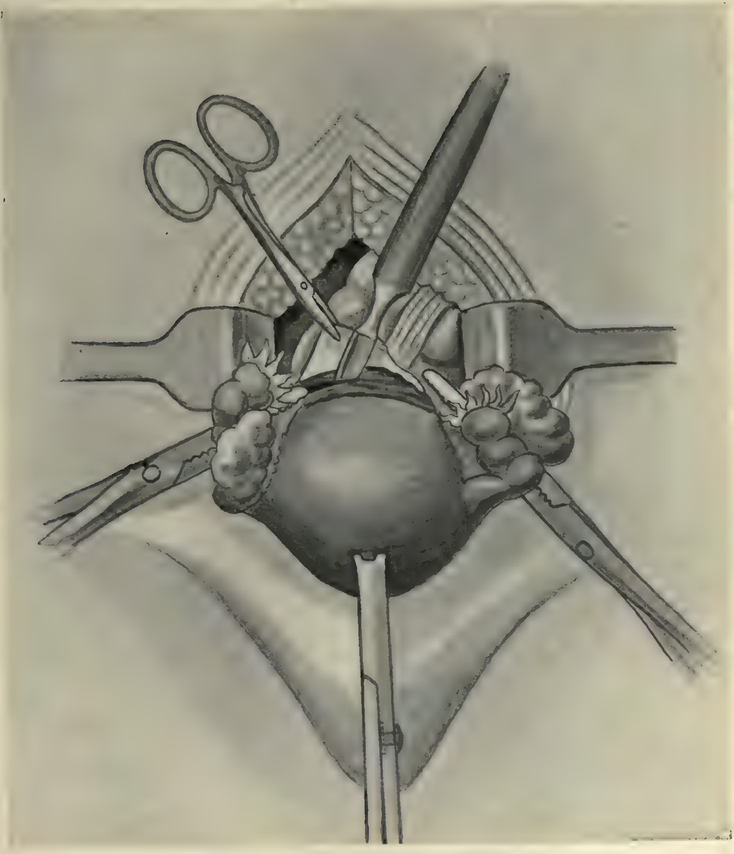


Fig. 33.—Murphy's method of doing a hysterectomy. Showing posterior incision through the corporocervical junction.

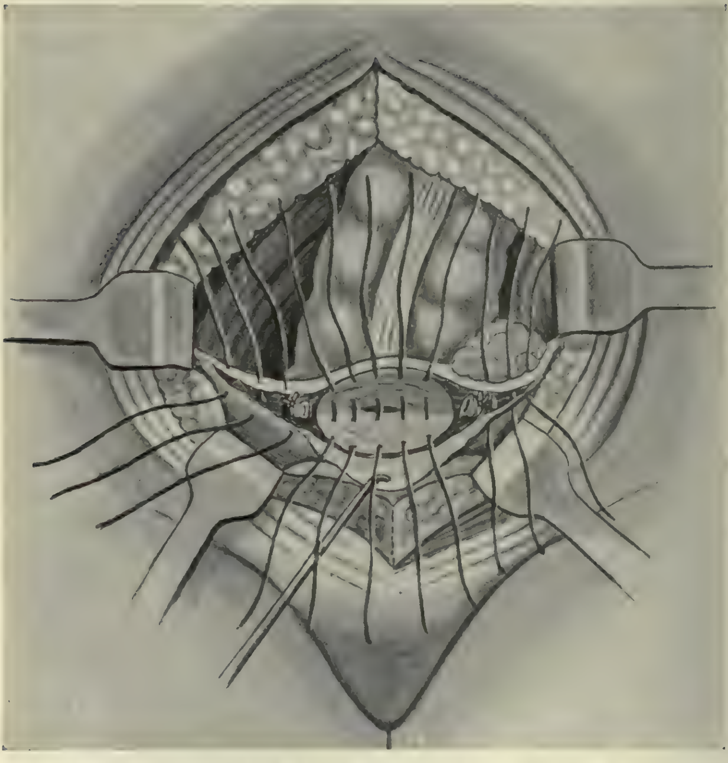


Fig. 34.—Murphy's method of doing a hysterectomy. Showing the cervical sutures and broad ligament in place.

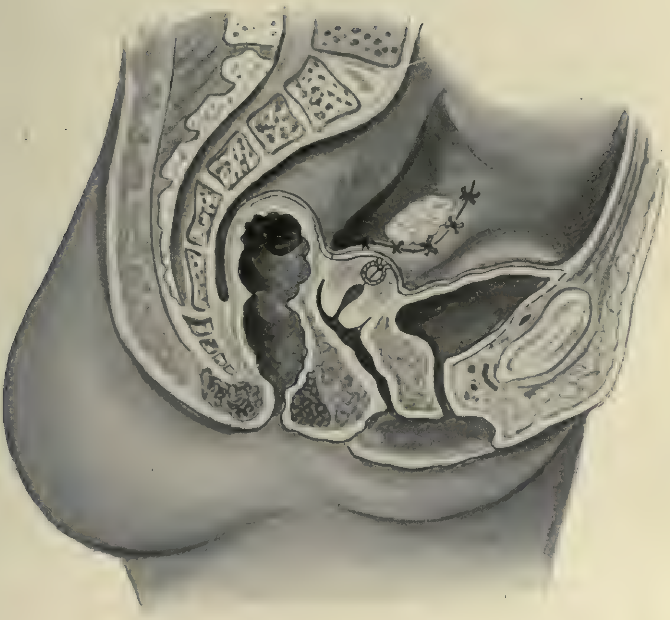


Fig. 35.—Murphy's method of doing a hysterectomy. Showing the cervical sutures tied and abraded surface covered with peritoneum.

is never made through the linea alba, and no muscle-fibers are cut.

4. Examination of the Pelvic Organs.—The uterus, ovaries, and tubes are carefully examined, to determine the exact nature and extent of disease adhesions and the line of procedure to be followed.

5. Protection of the General Peritoneal Cavity and Displacement of the Intestines Out of the Field of Operation.—This is accomplished by means of laparotomy pads.

6. Separation of adhesions, if any exist, between the uterus and surrounding structures. If the omentum is adherent, it is ligated close to the uterus and divided. Intestinal adhesions are separated by blunt dissection, care being taken always to work in the direction of the adherent coil, thereby adding to the thickness of the intestinal wall and avoiding perforation.

7. Securing Control of the Uterus.—For this purpose we use a large volsellum forceps or a corkscrew, which is inserted deep into the upper portion of the myomatous uterus.

8. Clamping the Broad Ligaments.—The uterus is drawn out through the abdominal incision with its posterior surface uppermost. Long heavy hysterectomy clamps are now applied to the broad ligaments close to the sides of the uterus, the ends of the blades extending down to the corporocervical junction, but not including the uterine arteries. There is no danger of injuring the ureters in this step of the operation if care be taken to place the ends of the clamps in direct contact with the uterus, above the level of the arteries.

If the tubes are diseased, they are removed with the uterus by dividing the mesosalpinx of each before applying the clamps to the ligaments. If healthy, however, their uterine ends may be included in the grasp of the forceps and both tubes allowed to remain. In patients who have not reached the menopause, the ovaries, or at least one of them, should always be retained. This is absolutely essential to the future welfare of the patient, and is nearly always possible. Even when both ovaries are diseased, it is possible, by resection of the affected parts, to preserve a portion of one or of both.

9. Division of the Broad Ligaments.—The ligaments are divided with the scissors ¼ inch to the inner side of the clamps, and the uterus, which is now liberated from its lateral attachments, and is rotated downward and forward. This brings the posterior surface well into the field of operation.

10. Incision Through the Corporocervical Junction.—A transverse incision is made with the scalpel into the posterior wall of the uterus at the corporocervical junction, and the cut edge of the peritoneum is secured with artery forceps. This incision is directed forward and slightly downward as far as the cervical canal, and then a little upward and forward toward the bladder until two-thirds of the anterior portion of the corporo-cervical muscularis is divided. The volsellum is then placed on the cervix and firm traction made. The uterine arteries peel away from the muscularis and are easily seen on either side of the cervix. The uterine arteries now come into view on each side, and are secured with artery forceps before they are cut. Should they not be plainly visible, however, no time is spent in looking for them. From the level of the canal the incision is continued through the cervical tissue, the operator drawing the uterus forward as he proceeds and rolling it away from the anterior peritoneum and bladder. In this way a peritoneal flap is formed which is sufficiently large to cover the cervical stump. If the uterine arteries are not clamped before they are divided, the assistant grasps them with hemostats when they begin to bleed, while the operator continues his incision. Once the uterine artery is exposed on either side, no further cutting in a lateral direction should be done, as the ureter always rests just to the outer side of the uterine artery. The artery is a sign-post that one is close to the ureter.

11. Ligation of the Uterine Arteries.—Each uterine artery is ligated with No. 2 plain catgut, and the hemostats are then removed.

12. Suture of the Cervix.—The wedge-shaped gap in the cervix is closed with interrupted catgut sutures, which approximate the cut surfaces but do not include the peritoneum.

13. Treatment of the Broad Ligament Stumps.—The broad ligament stumps may be treated in two ways: (*a*) By ligation *en* *masse*, which is exceptional with us. (*b*) By ligation of the individual vessels. The latter method we consider preferable. When the mass ligature is used, it should be tied in the crease produced by the clamp, for the following reasons:

(1) The compression of the clamp forces out all the fatty and areolar tissue, leaving nothing but the vessels and peritoneum within its grasp.

(2) The clamp acts as an angiotribe by injuring the intima of vessels and thereby favoring clot formation within their lumina.

(3) The ridge of tissue between the crease and the cut edge of the ligament prevents the ligature from slipping.

While the mass ligature has given perfect satisfaction, we believe the better plan is to ligate the two individual vessels after the clamp has been removed.

14. Covering the Abraded Surfaces with Peritoneum.—Commencing with the broad ligament stump on one side, a purse-string suture of catgut is inserted around it and the stump buried beneath the peritoneum. The same suture is used as a continuous Lembert suture, to approximate the anterior vesico-uterine flap to the posterior edge of peritoneum. When the broad ligament stump on the opposite side is reached, it is buried in the same manner as the first. By this continuous stitch all abraded surfaces are completely buried and nothing is exposed but the line of suture.

15. Cleansing the Peritoneal Cavity.—Blood-clots are removed by dry sponging, and the laparotomy pads are counted as they are taken out.

16. Prevention of Adhesions to the Lines of Suture.—The sigmoid is turned down and placed over the line of suture, in order to prevent the omentum from becoming adherent in the pelvis. This is of the greatest importance in all pelvic operations, as the omentum, fixed in this situation, may give rise to much suffering afterward. A case recently operated may be cited as an illustration. The patient had undergone a double oöphorectomy ten years ago for some pelvic disorder. She recovered from the operation, but soon afterward began to suffer constant abdominal pain, and gradually developed symptoms of dilatation of the stomach. At the second operation enormous gastrectasis was found; the pyloric zone of the stomach was adherent to the stump of the left ovary and tube.

17. Closure of the Abdominal Incision.—After drawing the omentum over the small intestine, the abdomen is closed by suturing separately the peritoneum (making the usual ectropion of its cut edges), fascia of the rectus, and skin. Heavy catgut is used for the buried sutures, and horsehair for the skin. Figure-of-8 silkworm-gut sutures are then inserted through the skin and fascia, to insure against separation of the wound in case the catgut is prematurely absorbed, and to obliterate dead spaces. Under the figure-of-8 stitch is placed a small gauze sponge to act as a buffer, preventing transverse necrosis of the skin.

When the patient is returned to her room, she is placed in a semi-sitting position, and, if at all depressed, is given two pints of normal saline solution per rectum every two hours. These enemata are given slowly, so that the fluid is absorbed almost as rapidly as it enters the bowel. The skin sutures are removed on the tenth or twelfth day, and the patient is allowed out of bed at the end of two and a half weeks.

If the fibroid extends into the broad ligament, the incision of its capsule should be made on the posterior surface, and the tumor enucleated through the posterior wall of the ligament.

The advantages of the Murphy method may be summed up as follows:

1. The tumor and uterus can be removed about as readily and as rapidly as an ordinary ovarian cyst, the average time required for the entire operation being from fifteen to thirty minutes. Most of the time is consumed in covering the abraded surfaces with peritoneum.

2. The danger of injury to the ureters is reduced to the minimum by rolling, instead of cutting, the uterus out of the surrounding connective tissue, thus following the natural lines of cleavage.

3. There is practically no danger of secondary hemorrhage, as each vessel is ligated separately.

The covering of the stump part of the operation was done by

Billroth when I was in Europe in 1882. The only difference between this technic and the old way is that in the Billroth operation the uterine artery was not tied separately but *en masse.* That hysterectomy differs from the Kelly hysterectomy in that Kelly cuts down in front and exposes the uterine artery or splits the uterus. I did not have to find the ureter in this case, because when I located the uterine artery I knew where the ureter was. If you get the uterine artery, you always know where the ureter is. There was no time in the operation when I was not absolute master of the situation, and especially so far as control of hemorrhage was concerned. By placing the sutures through the entire thickness of the uterine wall I stopped any oozing that might come from the cervical portion of the uterine artery. When placing the last row of stitches, the needle is not passed through the uterus at all, only through the serosa. The operation is a simple one, and very effectively controls hemorrhage. Furthermore, it insures against post-operative hemorrhage.

We said that this seemed to be a case of essential uterine hemorrhage without any change in the mucosa. There is another condition of the uterus which is accompanied by hemorrhage, and that is an essential uterine fibrosis. That condition demands a hysterectomy, much the same as does a fibroid.

You will note that in doing this operation we used just two ligatures—one on each uterine artery. I remember my visit to Edinburgh in 1883, when I called on the elder Keith, just after a stay in Vienna. The first question Keith asked me was: What is Billroth doing with his fibroid stumps after a hysterectomy? What did that mean? It meant that he had been having hemorrhages, notwithstanding the fact that he had at that time surprised the profession by reporting 68 consecutive laparotomies for ovarian tumors without a single death. I told him that Billroth was dropping the stumps back into the cavity. We are treating the cases in the same way today, except that we use only two ligatures, one on each uterine artery, and we tie them just as we tie the radial artery after an amputation of the arm.

This case is typical of a pathologic condition of which we know nothing, but a clinical history of which we know much. The hemoglobin percentage is 65. That is a point I want to emphasize. She looks anemic—has all the appearances of a secondary anemia from her last hemorrhage. The examination showed the uterus to be a little larger than normal, but perfectly mobile. The question came up as to what was the matter with her. The menstrual history differs from the menstrual history of a carcinoma. There is no gross pathology on the surface of the uterine mucosa nor in its wall to account for the hemorrhage. The hemorrhages extended over a period of about eight years—from 1904 to the present time. If she had had a fibroid there would have been an increase in the flow with each menstruation, except in the case of the subserous or subperitoneal type of tumor.

We came to these conclusions. Did she have a retained portion of the placenta from her miscarriage? Occasionally that occurs, but the rule is that these patients regularly bleed very much more at their menstrual periods. Did she have a deciduoma? A deciduoma runs a malignant course in a very short period of time, so that we excluded both of these conditions. What was it? I said I thought it was a case of essential hemorrhage of the uterus. Essential hemorrhage of the uterus is allied to the fibrosis of the uterus that occurs often in young people, and at the menstrual flow the uterus bleeds just like a sponge. It is often associated with fibrosis. I have seen four of these cases. In one I did a vaginal hysterectomy without an anesthetic, so near death was the patient. She had had nine children in rapid succession, and this bleeding had been going on for a considerable time. She recovered. That was about twenty years ago, and she is still alive and well. Following that, I had one case in a young woman, and following that I had one in a married woman who had had six children in rapid succession. She had uterine hemorrhages for which her doctor packed the uterus. We made a direct transfusion from her brother. Her condition improved temporarily. She finally showed evidences of hemolysis, became slightly icteric,—that lemon yellow tint that is seen in pernicious anemia,—and died at the end of four weeks. We did not remove the uterus. I thought that she had a deciduoma, and made a diagnosis accordingly. We held a postmortem on her and found that the surface of the uterus was perfectly smooth, and that there were no lesions in the uterus. That uterus was perfectly normal in every respect, but she had a small tumor the size of a hen's egg in each ovary. The pathologists could not agree on the diagnosis; some believed they were sarcomata while others believed they were not malignant.

There is nothing on the mucous surface of this uterus to account for this bleeding. The uterine wall is very fibrous but not much thickened. There are several pin-head-sized masses under the serosa. Whether the lesion in the uterus had anything to do with that woman's condition I do not know. We made a diagnosis of essential uterine hemorrhage and advised a hysterectomy. I was asked whether a curetment would not do just as well. I said "No," because she would bleed more after a curetment than before, as there was no endometritis, edematous or granulation condition of the mucosa of the uterus. I performed this operation because I believed there was a pathologic lesion present which demanded a hysterectomy. It is possible that essential uterine hemorrhage is one of the types of visceral hemorrhage or hemophilia described by Senator as occurring in the kidney. ("Renal Hemophilia," Berl. klin. Wochenschr., 1889.)

[The subsequent course of the case was uneventful. The patient left the hospital four weeks after the operation, feeling entirely well.—Ed.]

PYLORIC ULCER, WITH ENORMOUS HYPERTROPHY OF THE STOMACH MUSCLE

HISTORY

Patient, a male, aged thirty-eight, enters hospital on account of inability to retain solid food. Present trouble began fifteen years ago, when he experienced some distress after eating. This continued for several months; then he began to have acute, sharp pain in the epigastric region, about one-half hour after eating solid food. He would become slightly nauseated, and vomit about three-quarters to two hours after eating. Following the vomiting the pain subsided, but a slight soreness in the epigastrium continued to distress him. The vomitus consisted of the food ingested, was very sour, and contained no visible blood. The patient became unable to retain any solid food, and pain, nausea, and vomiting always followed on the ingestion of solid food. This continued for three years, during which time the patient lost 60 pounds in weight. About eleven years ago (three years following the onset of the trouble) patient avoided solid foods and began to use malted milk and eggs. Since then he has been almost free from pain, but the slightest variation from his usual diet is followed by sharp epigastric pains and then vomiting. Patient also complains of abdominal distress due to distention. This occurs two hours after eating, especially after the evening meal. If he lies on his right side, he is awakened by a sense of fulness in the abdomen, and then belching follows. Relief is obtained by lying on the left side. Occasionally he experiences pain after his customary liquid diet, but he seldom vomits. Has never noticed blood in either vomitus or feces.

COMMENTS AND OPERATION

Dr. Murphy: The examination shows that it is not a dilated stomach which gave him the distress; nor has he the severe pain which accompanies an acute ulcer. The means we use to examine the stomach is to give the patient a double Seidlitz powder. When we give that, we always have the stomach-tube and a basin ready, and if the patient has severe pain, we immediately pass the stomach-tube so as to avoid rupture of the stomach. There are cases reported in the literature of the stomach bursting after the ingestion of bismuth as well as the use of a Seidlitz powder. We lessen the likelihood of that accident occurring because in cases of acute gastric ulcer the sudden distention of the stomach always causes severe pain,—a stabbing pain,—and if the stomach-tube is passed immediately, the gas can escape from the stomach, the distention is relieved, and rupture is not so likely to occur.

The history of this case is very interesting, Fifteen years have elapsed since this condition began. For eleven of the fifteen years he has been subsisting on an egg and malted milk diet. Whenever he deviated from that diet, he immediately had all the symptoms of gastric obstruction. If this patient had an ordinary case of obstruction of the pylorus, we would expect to find a dilated stomach if he had been living in a natural way. But he has not been living in a natural way: he has been living in an unnatural way, on eggs and malted milk. The skiagram shows that the entire stomach is to the right of the median line. A peculiar thing about this case is that about sixteen hours after the bismuth was given to make the skiagram it was vomited as a semi-solid bismuth paste, showing that there had been retention of the solid portion of the bismuth meal in the stomach. The bismuth had not passed through the pylorus into the intestine. The fluid portion of the meal escaped from the stomach through the pylorus. The result would have been the same if solid food had been taken. Liquids or soft foods evidently pass through the pylorus. That is a very interesting case.

After the first attack he lost about 60 pounds in weight, but he has regained almost his normal weight. One would not think, from looking at him now, that he has a large ulcer in his pylorus, showing how well nourished one can be even on such a restricted diet. Of course, the history is the history of an ulcer. Whether it is a pyloric ulcer, or an hour-glass contraction the

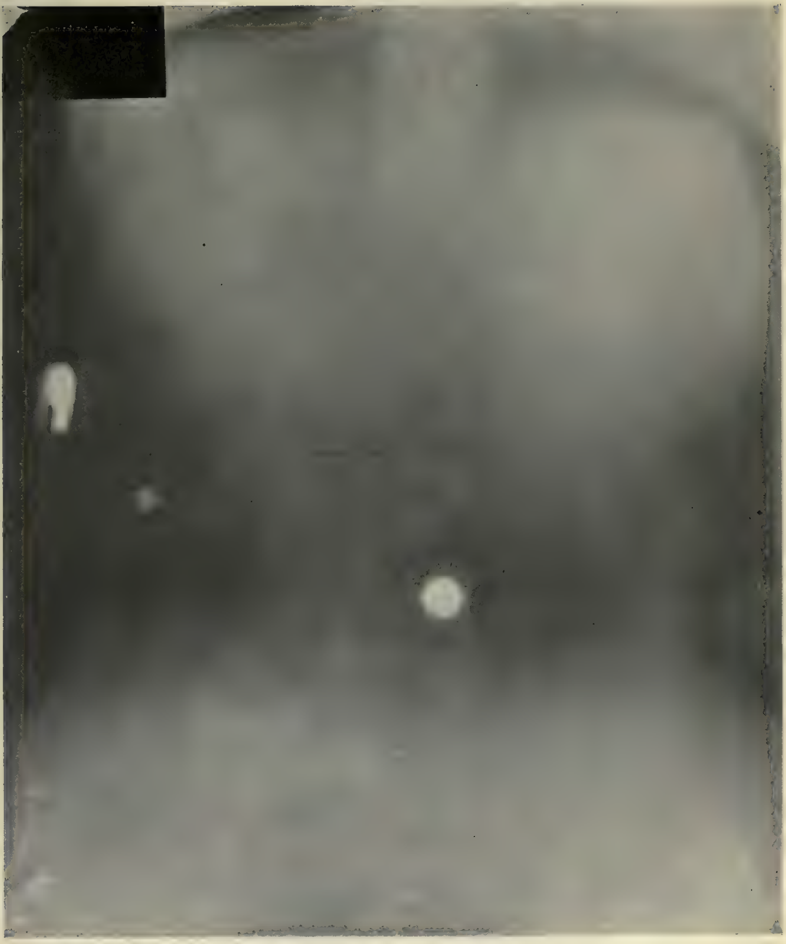


Fig. 36.—Pyloric ulcer. Five minutes after ingestion of bismuth meal. The large white area to the right of the spinal column is the dilated stomach. The darker shadow over the spinal column to the left of the stomach is the pylorus. No bismuth is passing.

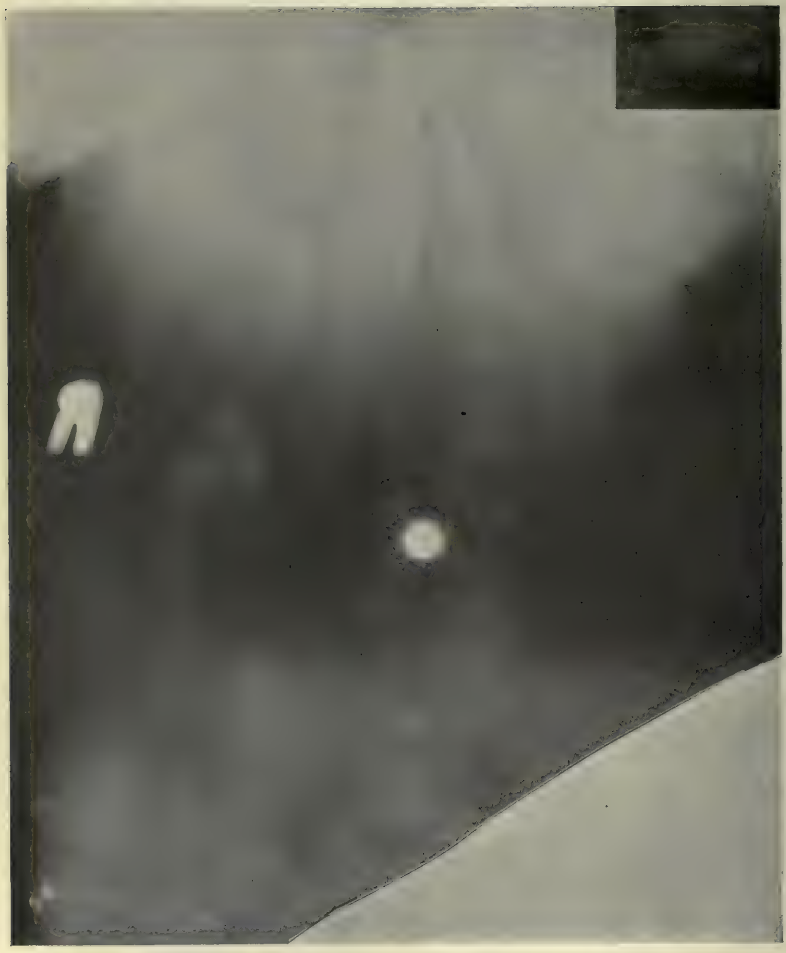


Fig. 37.—Pyloric ulcer. Four hours after the ingestion of the bismuth meal. The stomach is slowly emptying itself into the duodenum. The light twisted area to the left is the bismuth in the bowel.

result of the ulcer, I am unable to say. The pylorus is on the right side, but there is no secondary pocket, as one would expect to find in an hour-glass contraction of the stomach. But there is really no need of a secondary pocket, as the solid food all stays in the first pocket. Here is a case where the skiagram must be used merely to confirm the clinical diagnosis. It is not going to clear up the stomach lesions: it is merely an aid to the usual clinical methods of examination. It is of great value only in a small percentage of the cases. We are now using the Röntgen ray in a great many of our stomach cases. However, one must always resort to laboratory examinations, or, as we call them, instruments of precision. However, all of these are only adjuncts of the clinical examination.

This man has had all the symptoms of a gastric ulcer. He had epigastric pain, which was increased by eating and relieved by vomiting. The pain came on usually one-half hour after meals, diminishing in severity as time progressed. It appears to be a case of epigastric discomfort, with a great loss of weight during the acute attack. Then there are all the manifestations of an obstruction of the pylorus. There is the pain coming on at a time when the process of digestion should be nearly completed. The stomach, in place of emptying itself through the pylorus, emptied itself through the esophagus. He vomited. He has been eleven years on a malted milk and egg diet. I asked him the other day, "What would happen if you were to eat a meal of corned beef and cabbage?" He said that he would have distress and pain for four or five hours until all the food ingested would be vomited. A large quantity of the fluid food passes through into the intestines, but the solid part does not. That is shown by the skiagram. From the skiagram we are unable to make the differential diagnosis as to whether this is an hour-glass contraction or a pyloric block. The bismuth did not pass out of the first pocket. There does not seem to be a second pocket.

We will expose the stomach through the usual right rectus incision. There is a little dilatation of the duodenum, but not much. The stomach is enlarged and deep red in color. The condition present fits the clinical course, and it is on the clinical course that we base the diagnosis. This is not a dilatation of the stomach, but an enormous hypertrophy of its walls, especially the muscular layers. The stomach looks like a biceps. That extraordinary muscle development is the result of the pyloric obstruction, from which he has suffered for fifteen years. There is a scar of an old healed ulcer on the pylorus, which produced a decided contraction of the pylorus. The stomach is as active as any voluntary muscle; in fact, it is difficult to hold it in the grasp of the fingers.

The gall-bladder is also large, but does not seem to be pathologic in any way. We will do a posterior gastro-enterostomy in this case, using our oblong button. (For the technic of this operation, see the operation described on p. 212.)

Visiting Doctor: How long does the button remain in?

Dr. Murphy: We look for it to pass in about eleven days. Let the record show that the pathologic findings corresponded exactly with the clinical course; that there had been an old ulcer in the pyloric zone of the stomach that had produced a contraction of the pylorus for fully an inch, making a tube of muscular and cicatricial tissue, the opening in the tube being as large as the end of a lead-pencil. There were no adhesions, showing that it was a hypertrophic ulcer.

Let the record further show that the muscularis of the stomach was enormously developed, so that it was difficult to hold it in any position on account of its intense muscular contraction due to the effort of the stomach to overcome the contraction of the pylorus. There was no perforation of the stomach.

He never vomited a large quantity of fluid, showing how the stomach took care of liquids. We say in our text-books that liquids have to go into the intestine before they are absorbed. We gave him a bismuth meal and the skiagram showed that the bismuth was retained in the stomach. Sixteen hours after he was given the bismuth meal he vomited a bismuth paste. The fluid had disappeared. The same thing would have occurred if he had been given corned beef and cabbage. The clinical history and the clinical findings correspond. We believe in the interpretation of the clinical conditions and physical signs into pathologic entities in our diagnosis.

We did not believe that it was an hour-glass contraction of the stomach, because we were not able to find a second pocket of bismuth. All the bismuth remained in the first pocket.

In doing the posterior gastro-enterostomy the Germans are divided into two schools. At the last meeting of the Deutsche Chirurgische Gesellschaft, only 25 per cent. of those present stated that they performed the anterior operation. The larger number did the posterior operation. A smaller percentage perform a lateral anastomosis, doing the Roux operation or some other type of enteric anastomosis. They claim that by doing so they do not have the vicious circle. I think there were probably four out of the ten or fourteen present who declared they ever had the vicious circle. They reported a large number of cases—about 2500. They were divided as to whether they should do the anterior or the posterior operation. A small number went through the gastrocolic omentum and did a postero-anterior anastomosis, that is, they bring the small intestine up behind the colon and well up on the anterior wall of the stomach. In the later records of the cases it appeared that several of them had gangrene of the colon. A still larger number of them did the operation of gastro-enterostomy when the ulcer was high up on the lesser curvature of the stomach. The majority of the operators do the posterior, no-loop operation either with or without the oblong button. In Czerny's clinic at Heidelberg the button has been used for a longer period of time and more persistently than in any other clinic, and it is resorted to almost uniformly at the present time (Caan). In cases of ulcer some distance from the pylorus that does not interfere with the outlet of the food, we do not do a gastro-enterostomy because we have had so many cases that were not improved by it. We had a very interesting case recently in which we did three laparotomies, and each time we found a new ulcer higher up on the lesser curvature, with a perfectly patent gastro-enterostomy opening. After the third operation the patient got well and has remained well. It was a hard case to manage. [Button could not be located with *x*-ray when patient left hospital; therefore it must have been passed unnoticed.—Ed.]

DUODENAL BLOCK

Result of Adhesions Following Attempted Perforation of Ulcer

DISCUSSION OF MEDICAL PHASES BY DR. C. L. MIX

HISTORY

PATIENT, a male, aged thirty-three, enters hospital on account of severe pain in the epigastric region. Present trouble began about fifteen years ago, with a dull aching pain in the epigastric region. This attack had no relation to the kind of food or time it was taken. Patient felt nauseated, but did not vomit. This pain continued for two or three days, then ceased abruptly. These attacks came on about once a month for three years, when they became more severe and of longer duration. Sometimes the attacks lasted two weeks. Similar attacks have occurred up to the present, and have recurred about five or six times a year. Has vomited only twice—some years ago. Never vomited blood. Last attack began on November 20th and ended December 10th—about a month ago. During this time the patient had a sharp, cutting pain in the epigastrium, alternating with a continuous dull aching pain. Did not vomit, but felt nauseated for several days. Tenderness is present in the epigastrium during the attack. Has never had any chills or fever, nor has he ever been jaundiced.

COMMENTS AND OPERATION

DR. MURPHY: How about the operations he had?

INTERN: He had one for appendicitis three years ago.

DR. MURPHY: What was the striking fact about that operation?

INTERN: Within a week after the operation he had another attack.

Dr. Murphy: Yes, but that is not mentioned in the history. The important thing is missing. One might interpret this as an attack of appendicitis, but this man never had an attack of appendicitis. There was no evidence at the time of the operation to account for the symptoms, and he had recurrent attacks of pain after the operation, the same as before. He vomited only two or three times. What about the feces?

Intern: There was no blood in the feces.

Dr. Murphy: What was the blood-count?

Intern: White cells, 7800; red, 4,200,000; hemoglobin, 80 per cent.

Dr. Murphy: This man has a history extending over about fifteen years, but the attacks have become more frequent of late. There is no blood in the urine. The lesions one has to differentiate in this case are:

First: An appendiceal lesion, but that is eliminated by the fact that he has had his appendix removed.

Second: An obstruction to the ureter. With that condition the patient has pain in the loin radiating down to the glans penis, which is absent in this case, as are all the other symptoms of ureteral block.

Third: Duodenal ulcer. He is neither relieved nor distressed by food, which is an interesting fact in this case, and he has not had any blood in his feces.

Fourth: Intestinal retention that occurs in connection with adhesions that are formed after a previous operation. He cannot have a pyloric obstruction, for if he had, he should have gastric distress and vomiting. Occasionally, the pain is caused by a megaduodenum. That is due to a compression of the superior mesenteric artery. That compression occasionally produces a retention in the duodenum. This was the duodenal condition that was recognized by Rokitansky, and I think it was Codman, of Boston, who recently made some experiments on the cadaver, showing that there is a retention of the intestinal contents in the duodenum, due to compression of the superior mesenteric artery. He failed, however, to take into consideration the fact that the mesentery in the cadaver is thick and never elastic, whereas the

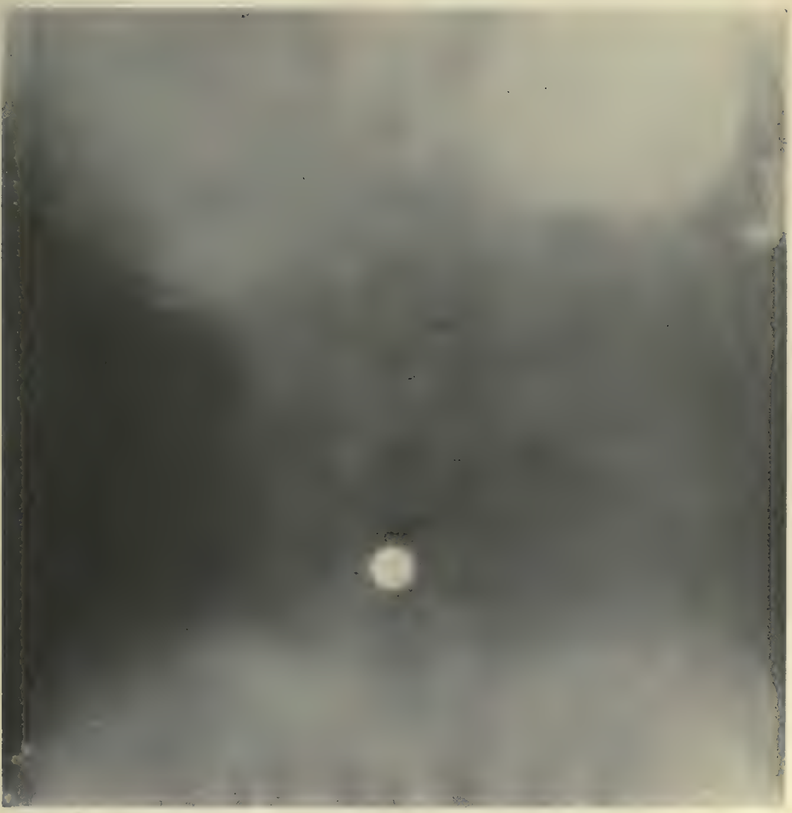


Fig. 38.—Duodenal block. (Positive made from negative.) Skiagram made five minutes after ingestion of bismuth meal. The large white area on the right is the stomach. Leading from the left of the stomach is a narrow white channel, indicating the narrowed pylorus. The white, bulb-shaped area into which this leads is the duodenum.

Fig. 39.—Duodenal block. (Positive made from negative.) Skiagram made two hours after ingestion of the bismuth meal. The stomach shadow is much smaller, the narrow line is much longer, and the bismuth (white areas) is seen in the intestines.

mesentery in the living is not thick and is elastic. There are no symptoms of mechanical ileus in this case, or of intestinal adhesions without ileus.

The first skiagram made shows that there is a retention of a considerable quantity of the bismuth in the duodenum. This man had the splash that occasionally is heard in cases of duodenal obstruction. That splash also occurs in the hour-glass stomach and in megaduodenum. The stomach is in its normal position, on the left side. There is a narrow band crossing over the dilated duodenum on the right side. In the second skiagram the stomach is almost empty, but the duodenum is not. The duodenum ought to be empty. After all is said and done there are not symptoms enough present in this case to make a differential diagnosis. Dr. Mix made a diagnosis of duodenal block in this case.

Dr. Mix, will you discuss the case?

Dr. Mix: The patient's trouble began fifteen years ago, and has continued with varying intensity up to the present time. There were only two positive statements of diagnostic value in the patient's history. The first of these is that the pain, when he has it, is in the epigastrium; that it is usually a dull aching pain, but that at the time of the attacks it is severe. The other positive statement which he makes is that these attacks have occurred for eighteen years—about five or six times in the course of each year.

There is, furthermore, in the history an exceptionally important point, namely, an operation for appendicitis three years ago. The patient made a statement to me that within five days after his appendix had been removed he had an attack of the same old familiar pain. Evidently the patient never had an appendicitis. Possibly a diagnosis was made of a chronic appendicitis with a secondary pyloric spasm, to account for his symptomatology.

There are also in his history some negative points of positive value. The first of these is the absence of nausea. It is true that some years ago he vomited twice, but all of us have vomited at some time or other. He also states that he never vomited blood, and has informed me that, to the best of his knowledge and belief, he never passed a bloody stool. Another statement which may be of some value or may be a stumbling-block in the diagnosis is that there is no diminution of the pain on taking food; neither has there been any increase of the pain when he eats. His last attack lasted from November 20th to December 10th. In describing it he states that he had a severe cutting pain in his epigastrium, associated with nausea, some epigastric tenderness, but no fever, no chill, no jaundice. Such a history is colorless—it neither affirms nor denies the possible existence of gall-bladder disease or gastric or duodenal ulcer. It raises the presumption against a diagnosis of gall-bladder disease by reason of the absence of the signs of infection.

Physical examination gave us the key to the diagnosis in this case. Aside from the degree of epigastric tenderness, he had well-marked splashing. On passing the stomach-tube I took from his stomach two or three ounces of residual food four hours after his last meal. I then had given to him an Ewald test-meal and a motor meal. The Ewald test-meal gave us no blood and a slight degree of hyperchlorhydria. When the tube was passed for the motor meal, the stomach was found to contain no food. This tube was passed by the nurse, and I am unable to state whether at the time it was passed a splashing might not have been found in the duodenal area.

Since both the Ewald test-meal and the motor meal indicated to us the absence of pyloric blockade and since, notwithstanding the pyloric blockade there was evidence, by reason of the splashing, of food retention, we decided to give the patient 16 ounces of buttermilk containing 2 ounces of bismuth subcarbonate, and to take an *x*-ray plate immediately after the ingestion of the buttermilk and bismuth, and a second picture two hours later. The first *x*-ray plate, as already stated by Dr. Murphy, shows a path of retained bismuth in the duodenum. In all our skiagrams and *x*-rays we have not found such a duodenal picture unless a duodenal blockade has been present. The first plate (here it is!) does not fully contain the evidence of a duodenal blockade, but in conjunction with the second picture, taken two hours later, the evidence seems conclusive, because it shows here the same spot as in the first picture, and a somewhat smaller amount of retained bismuth. Therefore the conclusion seems to be inevitable that there is in the second portion of the duodenum a constriction. Such a constriction might be occasioned by a duodenal ulcer, but we have no evidence in the case, aside from the long continuation of his illness, of a duodenal ulcer. If he has a duodenal ulcer, it is certainly not active. Adhesions, secondary to gall-bladder disease, might also lead to the same result, but there is in his history nothing pointing, even with a finger of suspicion, at the gall-bladder. We, therefore, are forced to rest merely with the diagnosis of duodenal block.

Dr. Murphy: We will make the incision over the right rectus muscle, near the midline, going through the skin and muscle-sheath, then displacing the muscle-fibers outward. The peritoneum is grasped between two tissue forceps and cut on the slant. We are now in the peritoneal cavity, and will begin our examination. The gall-bladder shows evidences of a pericholecystitis. There are some adhesions here. I want to call your attention to that because of the fixation that has taken place of the gall-bladder to the pancreas. That gall-bladder ought to be perfectly free. An inflammatory adhesion has fixed it, however. I must separate that adhesion before I can get at the duodenal zone, which I wish to expose. That adhesion is pathologic. There is an infiltration here extending back to the pancreatic zone. The band of connective tissue around that gall-bladder is an evidence of the intensity of the inflammation that fixed these intestines and limited their mobility. There is no stone in the gall-bladder or common duct. The mesocolon is very thick, the result of previous inflammations.

I have succeeded in freeing the large intestine, so that I am able to palpate the gall-bladder with ease. It is easily emptied by finger pressure. Now we will examine the duodenum. It is fixed. I think there has been an ulcer there. There is an old healed ulcer, and, judging from the fixation of the bowel and the adhesions present here, the ulcer must have endeavored to perforate through to the pancreas. That accounts for all the pathology in this case.

The duodenum is blocked by these adhesions and infiltrations in the lower part of the descending loop, by an inflammatory band of connective tissue. There is the dilated portion, proximal to the band of adhesions, that we concluded was present from the splashing we heard. There is no ulcer of the stomach in this neighborhood.

Dr. Mix: The pain was in the duodenal area, and the pain had no relation to food or time of eating.

Dr. Murphy: The duodenal ulcer is still infiltrated, and has attempted to perforate into the pancreas. The pancreas is fixed for a distance of an inch. I will have to do a posterior gastro-enterostomy. The hard, firm rope of connective tissue constricted the duodenum, causing the block. Let the record show that the infiltration extends well back into the pancreatic zone; that there is no infiltration around the gall-bladder, but that there are old adhesions. There is no stone in the gall-bladder and no stone in the common duct. There is more pathology here than I expected to find.

There are some adhesions at the posterior wall of the stomach.

I see now the ligament of Treitz. We must always look for that ligament to know that we are in the right place, and to get our bearings. I pick up the highest loop of the jejunum, and then I am orientated. The omentum and transverse colon are then turned upward over the epigastrium, and held by an assistant so that the mesocolon is made taut. At a bloodless spot within the arch of the midcolic vessels the mesocolon is split and the opening made large enough so that the posterior wall of the stomach can be drawn through. The lowest part of the greater curvature is selected for the anastomosis. The colon is then dropped down and the stomach drawn through the opening. Three or four catgut sutures secure the stomach to the mesocolon. A curved gastro-enterostomy clamp is applied transversely to the protruding portion of the stomach.

The highest loop of the jejunum is then emptied of its contents by "milking" it, and a second clamp is placed on a segment of jejunum. The two clamps are laid side by side, and the field of operation protected by dry sponges. Parallel incisions, about 1½ inches in length, are made in each viscus, between two rows of Pagenstecher linen through-and-through stitches, both ends of each suture being threaded in straight intestinal needles. One half of an oblong Murphy button is placed into the stomach opening, and the other half into the intestinal opening. The sutures are drawn taut and tied. The halves of the button are approximated and pressed together firmly. A few Lembert stitches are placed in the serosa at equidistant intervals, so as to relieve traction on the line of button approximation. The clamps are removed, and the operation is complete. The operation is not a difficult one, and it insures against hemorrhage more than any other method of intestinal anastomosis.

Let the record show that the muscularis of the stomach was developed in excess on account of the obstruction in the duodenal zone which it had been endeavoring to overcome. Let the record further show that the diagnosis of duodenal block, as made by Dr. Mix, was confirmed, and that it was due to an ulcer in the duodenum with a cicatricial band across its upper surface; that the duodenum was fixed to the head of the pancreas; there was considerable thickening of the head of the pancreas; that a posterior gastro-enterostomy, no-loop operation was performed with the oblong button; that there was no evidence of infection of the gall-bladder.

The peritoneum is closed with catgut, turning out the cut edges. We have been in the colon bacillus zone; that is, we have had the stomach and intestines both open, and we use an aqueous 1 per cent. lysol solution to stimulate the reaction of the tissues against the microörganisms. If you get a few colon bacilli in the fatty tissue, there is going to be trouble. The wound in the abdominal wall is closed in the usual manner, the aponeurosis of the rectus muscle being sutured separately. Figure-of-8 silkworm-gut sutures and horsehair complete the closure. The wound is dusted with bismuth subiodid and sealed with collodion gauze. The usual dressing is put on, held in place with broad adhesive straps and an abdominal binder. No drain.

[The patient left the hospital four weeks after the operation in splendid condition. The stitches were removed on the twelfth day. Button was not passed before patient left hospital, but word was received later that it had been passed.—Ed.]

ACTIVE DUODENAL ULCER NEAR PYLORUS. BLEEDING ABOUT SEVEN HOURS BEFORE OPERATION—A FEW GERMAN STATISTICS ON THE BUTTON

HISTORY

Patient, male, aged thirty, enters hospital on account of excruciating pain in the epigastrium, midway between the umbilicus and the ensiform cartilage. Present trouble began in the fall of 1905, about eight years ago. At that time he complained of a gnawing sensation in the epigastric region, usually coming on about two hours after eating. This was accompanied by a feeling of abdominal uneasiness, but no vomiting followed. Patient noticed at this time that eating relieved the pain. These attacks occurred about every two weeks that winter; during the following summer the attacks occurred less frequently. In the fall of 1906 the intervals between the attacks were shortened and the epigastric distress more pronounced. About three years ago the pain became very severe, and came on more frequently at night. Patient would be awakened by severe epigastric pain, accompanied by nausea but no vomiting. Taking food would relieve the pain, therefore he would have food near at hand every night to obtain relief when the pain would come on. For the past few months the pain has followed nearly every meal, and is becoming more intense with each attack.

About two weeks ago he noticed black, pasty, offensive feces in two successive bowel movements, but he has not noticed any change from normal since. About ten days ago he vomited at the height of the attack, and continued to vomit for about three days. Never noticed any blood in the vomitus. Last attack occurred January 17, 1913 (yesterday), about 1.30 P. M., two hours after eating. The pain was excruciating, and lasted five hours; it was accompanied by nausea and marked epigastric tenderness and rigidity, but no vomiting. Has never been jaundiced. Has lost 30 pounds in weight in the last year. Had appendicitis twelve years ago. Appendix removed. Neisseria infection five or six six times.

COMMENTS AND OPERATION

Dr. Murphy: This case has been gone over carefully by Dr. Mix. I will ask him to analyze the history.

Dr. Mix: Attention is called to this fact, that twelve years ago, when the patient was eighteen years of age, he had an attack of appendicitis and his appendix was removed. He says that no drainage-tube was inserted, but the scar is a ragged one. He insists that this is entirely due to the fact that while it was healing the dressings were pulled off with some degree of force and that the skin came apart with the dressings. This attack of appendicitis eliminates from our consideration any possible consequence of appendix disease, except, possibly, the post-operative adhesions.

The first salient point in this history is its duration, the onset occurring eight years ago. Its course has been intermittent, with periods of freedom from suffering alternating with attacks of epigastric pain, sometimes excruciating and sometimes consisting only of an annoying sensation. At times the localization of his illness has been vague; he describes it as extending over the whole abdomen. Though the pain was described in the intern's history as being in his epigastrium, the painful area lies a little to the right of the epigastrium and above the umbilicus, in the duodenal area, a little nearer to the pylorus than in most cases of duodenal ulcer. We found by cross-examination last evening, when I examined him, that he had more trouble in the spring and fall of the year than he did during the summer or winter. About this time of the year he ordinarily begins to get well, and again in the summer he feels comparatively comfortable, but in the early spring and in the early fall he feels decidedly worse. This symptom, which was so emphasized by Moynihan and Mayo in many cases of duodenal ulcer, raises the presumption in favor of that diagnosis.

Another important point in the history of this case is the relationship of the pain to the taking of food. The majority of cases of duodenal ulcer have pain coming on some time after eating, and the pain is relieved by the further ingestion of food. It is a probable fact that the hyperchlorhydria, which is almost always associated with duodenal ulcer, is responsible for the acid stream of hydrochloric acid bathing the ulcer as it flows from the pylorus, thus causing pain; it is a fact, further, that by the ingestion of food the pylorus is temporarily closed and the duodenal alkaline secretion temporarily increased, with the result that such acid as there is in the duodenum is neutralized at once and so the pain is obviated. I asked the patient if he was not one of those who gets up in the night to eat, and learned that he was a "night-feeder." Night-feeding is a very common symptom in these cases, many of the patients going to bed with some crackers or a glass of milk by their bedside.

A negative point of some importance is the absence of vomiting during the eight years of his illness. It is true that he vomited on one occasion recently during an acute exacerbation of his distress, but during the eight years he did not vomit at all, according to the history. About ten days ago, at the height of one of his attacks, he vomited for about three days. This is the only attack of vomiting that we have a record of in his history. Such vomiting raises a strong presumption of peritoneal involvement.

Also important is the question as to the presence of blood in the feces. During his stay in the hospital we have not found blood, but about two weeks ago, as I learned by careful cross-examination, for two days he had very dark stools. He had not been taking either bismuth or iron. The stools were not formed, but were mushy and pasty. He said that they "stank." Decomposed blood has a very foul odor. Such a history of black, pasty, stinking stools, two in number, being succeeded by normal stools, is very strongly suggestive of the presence of blood. There is no history of the vomiting of blood.

Important also are the negative symptoms, the absence of attacks of colic, jaundice, fever, and chills.

Physical examination shows very little. The only finding of importance is the tenderness over the duodenal area, near the epigastrium. This marks the location of the duodenal ulcer. It is near the pylorus, and evidently on the duodenal side. He has no icterus, therefore it does not lie near the duodenal papilla, or, if it does, does not involve it, and so produce an icterus. The ulcer is not on the gastric side, because the period of time (two hours) elapsing between the ingestion of food and the onset of the pain is too great, and also because of the absence of the vomiting of blood and absence of blood in the Ewald test-meal.

We are forced to the conclusion that he has a duodenal ulcer lying near the pylorus, and of eight years' duration. We did not find, on examination, any signs of pericholecystitis. There are no tenderness on pressure over the gall-bladder and no gall-bladder disease, so far as I can make out, either from the physical signs or the history of the case.

Dr. Murphy: This man had an attack of pain last night, which is exceptional for duodenal ulcer. The pain was so severe that he had to have a hypodermic of morphin. The pain was not general: it was confined to the epigastrium and a small area a little to the right. He had pain on the right side yesterday. We will make the usual incision, going in through the right rectus muscle, between the umbilicus and the ensiform cartilage.

Just as soon as the peritoneum is pinched he begins to stiffen. He appeared to be very much relaxed until we grasped the peritoneum with forceps; then he stiffened. We will draw the stomach out into the wound. There is no evidence of ulceration here. Running over the lesser curvature with my finger I find no induration. Now we will pass around to the pyloric zone. The pylorus does not come up as easily as usual. I can feel an ulcer on the posterior surface of the pylorus. It appears to be active. It is in an exceptional position for a gastric ulcer. There is a mass here that feels firms and hard, as though the ulcer might have perforated onto the head of the pancreas. The thickening is very pronounced. The mass is as large as a butternut, and hard as wood. If this mass were in the stomach, I would say at once that it is a case of carcinoma. Being in the duodenum, I can say that it is not a carcinoma; that it is an active ulcer, which accounts for the pain he had last night. It is, you see, a big, hard, and woody mass on the posterior wall of the duodenum. It is a condition which is very rare with duodenal ulcer. It looks more as if the ulcer had perforated clear through the wall of the bowel and into the head of the pancreas. The pain last night went right straight through to the back. It seems as if there were a mass in the pancreas, but on careful palpation I can feel only a general infiltration. In this case we must do a posterior gastro-enterostomy with the button. The chances are that this condition is not malignant, because it is in the duodenum. A malignant ulcer in that position is rare.

[The typical button anastomosis, side to side, was done, as described on page 212.—Ed.]

He has evidently had some bleeding—recent bleeding, probably last night. On opening the abdomen we find that the intestine is full of blood. That is very interesting, especially in view of the pain association. This is an exceptional case to see, because the acuteness of the pain last night resembled in its severity the type of pain that one has in a tuberculous ulcer just preceding the bleeding. This intestine is full of blood. The attack of pain last night was very severe.

I am delighted to see that blood in connection with that severe pain. That is an instructive case.

I recently read a very interesting article in the Archiv für Chirurgie. It was written by a surgeon who has been using the button exclusively since 1902 for his gastro-enterostomies, both of the anterior and posterior type. He reviewed the literature, which I have not done in years. I rarely talk about the button now, because I think that every one has found out for himself whether or not he wants to use it. He went through not only the recent literature, but all the literature in the clinic at Heidelberg. He mentioned a large number of cases, including those of Steinthal. The point that concerned me was how many failures with the suture and the button were due to hemorrhage.

There were 547 posterior gastro-enterostomies by Daniell, suture operations, with 14 vicious circles. In 458 cases done with the button he had 5 cases of vicious circle. In Heidelberg, out of 333 cases with the button, there were only 2 cases of vicious circle. In 447 suture operations there were 10 leakages, while in 397 button operations there were only 5 leakages or perforations, that is, about 1.5 per cent. One of these leakages was due to the suture. The surgeon put in the tacking stitch, and it went through to the inner coat of the stomach, which is exceptional. If he cut clear through the entire coat, that might easily have happened, but in putting in the tacking stitch in the right way,—a Lembert stitch,—you rarely get a leak.

The author's conclusions may be summed up in this expression: If you want to do a gastro-enterostomy and feel free from the danger of subsequent complications as regards leakage and hemorrhage, the thing to do is the posterior operation with the button. He says that the anastomosis done with the button is the ideal anastomosis; that no other operation gives such ideal unions. This was the title of my original article on the button—"An Ideal Anastomosis." Even in my dogs that line of union was so normal that it looked like a physiologic or anatomic connection.

I am merely giving the statistics as they are coming out now. This subject has been studied for twenty years. What I say for or against it does not matter to us a little bit. Czerny has been using the button ever since it came out, first for his desperate cases demanding hurried operations, mostly malignant cases, and now for all his gastro-enterostomies. Czerny is the father of the Czerny-Lembert stitch. We are not using it in all our cases. We do the suture operation in a small percentage of the cases, but we never feel quite secure as far as hemorrhage is concerned. Just recently we had a case in which there had been a gastro-enterostomy made by suture previously in which the opening had closed, leaving adhesions. We had to reform and suture the gastro-enterostomy opening at the point of leakage, which we did with a double row of the Czerny-Lembert type sutures. That evening she showed evidences of a severe hemorrhage. When we reopened the abdomen, we found that the patient had a gastric hemorrhage from the line of suture, and we had to resuture. We got a good result in that case. We gave her an intravenous transfusion on the table at the time of the second operation.

One point in using the button must be emphasized. In tying the puckering string around the neck of the button, always have the knot on the end of the cylinder, never on the side. It makes a much more secure approximation and helps to prevent leakage. Zielowicz charged me with a fatal case in the use of the button on account of that. The analysis of his report showed that the ends of the puckering-string stitch were outside the bite of the button. It was not the method, but the faulty technic which accounted for the death of this patient. This case was quoted as a fatality from the use of the button hundreds of times.

The history of this case is very interesting: the clinical course, the periods during which he would be perfectly well, as far as his gastric and duodenal symptoms were concerned; finally, the vomiting, and then this hemorrhage, which we found in the bowel today. He had previously had a melena. He had dark stools twice at times when he was not taking bismuth or iron, which must be interpreted that he had blood in his stools.

There were no stones in the gall-bladder. The acuteness of the attack is shown by the serous fluid that is present in the gastric, duodenal, and hepatic fossæ. Therefore we will put in a tube drain in this fossa. This drain will be removed in five or six days, when drainage probably will have ceased. The wound is closed in the usual manner—in layers; first, the peritoneum; second, the rectus fascia; third, the figure-of-8 suture, which obliterates dead spaces; and fourth, the horsehair, which approximates the skin edges. Bismuth subiodid, collodion gauze, a gauze and cotton dressing, adhesive fixation straps, and an abdominal binder complete the dressing.

[The patient made an uneventful recovery. Button was not passed before patient left hospital.—Ed.]

GASTRIC ULCER, ETC.

A talk delivered at Dr. Murphy's Clinic, January 22, 1913

By Mr. Robert Milne, F.R.C.S., of London, England

Dr. Murphy: We are very fortunate in having with us this morning a very celebrated surgeon, a man whom I met last year in the London Hospital. The afternoon that I called at the hospital he said to me: "We have here an acute gastric ulcer that has perforated within the last few hours, and upon which we are going to operate." Within two hours after the perforation he had the patient on the table and was closing up the pyloric opening. In that hospital on that day I saw five cases of gastric ulcer. The London Hospital has one of the largest services of this kind in the world, except possibly that of the Mayos.

Mr. Milne is one of the positively, thoroughly educated men who has had a wide experience. He is one of those men who, without any flattery, gives a plain statement of the facts. He is in that way one of the educators—one of the real educators—in medicine. He has very kindly consented to talk this morning on any subject he may desire.

Mr. Milne: *Dr. Murphy and Gentlemen:* If you will look at that list of 19 operations on the board, ranging from cerebral tumor down to tuberculosis of the metatarsus, you will appreciate my difficulty in choosing a subject, seeing that Dr. Murphy himself does work from the crown of the head to the sole of the foot. Since Dr. Murphy has mentioned peptic ulcers, I may put before you some points on these in connection with infections.

I believe the great essential in the treatment of any pathologic condition is to remedy the cause, if this be still active. Can we determine the cause of gastric ulcer? We know that in the mucous membrane of the stomach, and even partially in the submucous coat, there are small collections of lymphoid tissue. These are in greatest number along the lesser curvature, and in the prepyloric portion of the stomach; rare in the cardiac portion. Although small in a healthy stomach, when inflamed they swell up, push the mucous glands apart to each side, and reach the surface. Various terminations are now possible in such a swollen follicle:

First: It may simply resolve.

Second: It may break down in the center, and a minute bead of pus be discharged into the stomach.

Third: In the submucous coat there are the large blood-vessels. The follicular inflammation, if in contact with a vessel, may erode its wall, give rise to a miliary aneurysm, or finally lead to rupture of the vessel.

Fourth: The burst follicle, giving vent to the bead of pus, may allow the gastric juice to enter, to digest the coat of the stomach, and lead to the funnel-shaped, acute perforating ulcer to which, unfortunately, we have become accustomed. I shall enlarge on this later.

Fifth: The entry of gastric juice may lead to a chronic ulcer, and so later to carcinoma.

Terminations one and two would be called acute gastritis. Number three accounts for those cases of hematemesis in which gastrotomy revealed simply points oozing blood.

Termination four may seem too radical, for you will remember that several observers endeavored to produce ulcers by lacerating the gastric mucous membrane in animals. They were only rarely successful. So in the human stomach we believe that normally the edges of the mucous membrane fall together and gastric juice does not enter the wall of the stomach. If, however, from any cause, *e. g.*, fibrosis, due to repeated inflammatory attacks, the mucous membrane is altered so that the edges do not fall into contact, then the submucous coat is unprotected, and an ulcer follows from the penetration of the gastric juice.

Other experimenters injected lycopodium into the circulation to produce gastric ulcer. They were successful rarely. One pathologic condition seemed to support the theory upon which they were working—I refer to the "contact ulcer," the small ulcer which we may find on the wall of stomach or duodenum opposite a chronic ulcer. The theory would be that these might be terminal necroses due to embolism of a vessel on the lesser curve, just as blocking of the abdominal aorta might lead to gangrene of both legs, but not thighs. Pathologists started to look for these emboli. They could not distinguish them with the naked eye or with the microscope, and saw them only with the eye of faith. I believe these paired ulcers are not embolic at all, but that the second ulcer has been started by the infection of lymphoid follicles there from the septic surface of the primary ulcer.

We know how a pathologic appendix or gall-bladder will lead to pyloric spasm and gastric pain. We can go further. If you have in that gall-bladder or in that appendix an infective process, you have a possible source of infection of these lymphoid follicles in the stomach, and so a source of ulcer, and we have not done our duty to the patient in remedying the gastric ulcer unless, in addition to the operative treatment on the stomach, we remove that appendix, deal with that gall-bladder, etc.

Another pathologic lesion in the abdomen of interest to the surgeon and due to infection is cholelithiasis. How do the organisms reach the gall-bladder?

Mr. Bond, of Leicester, did some experiments. In patients with colostomies he found indigo-blue traveled from the rectum to the opening. He found in patients with vesical fistula that it traveled up the urethra and appeared in the fistula. The indigo-blue particles appeared in the bile from a gall-bladder fistula when administered by mouth. It reached the pelvic peritoneum, as shown in laparotomies for pelvic trouble when placed inside the cervix twenty-four hours before.

These phenomena were not due to capillary action, for they did not occur in the cadaver, therefore were vital. They were not due to reverse peristalsis, for a cathartic hastened their appearance in a colostomy. They were not due to ciliary action, for there are no cilia in the bile-ducts or urethra. Indigo-blue is a bit soluble in bile, but it is deposited from bile as crystals, while in these experiments it was deposited as amorphous granules on the wall of the gall-bladder. He explained the phenomena by ascending mucous currents on the walls of the ducts or tubes.

Notice you: All these experiments were on pathologic subjects and are not proof as to what would happen in a healthy subject. We know that if a man has an infection in the bladder, there is little risk of that infection going up the ureter to the kidney unless there is some obstruction to the ureter. We believe that infection in the gall-bladder comes not through the common bile-duct, but through the circulation, the organism having escaped the destructive ability of the liver-cells, being, however, as a rule, greatly attenuated in virulence. I had to operate on a woman aged seventy-three about one year ago for acute cholecystitis, and her case illustrates the long time organisms may live in the gall-bladder. She had suffered from typhoid forty-five years before; her gall-bladder gave a pure culture of typhoid bacilli; she gave a positive Widal, with a dilution of 1:132, and one of the nurses in charge of her developed typhoid, the patient being the only source we could discover.

A third complication of interest to the surgeon is in the kidney. After speaking of gall-stones, one would naturally think of the relation between infection and renal calculus. We have all, unfortunately, had to deal with patients coming with both kidneys packed with large dirt calculi and perhaps removed them. With what result? That man will come back to us for certain within two or three years with reformations as big as the former calculi: why? Because those stones were due to infection of the pelvis. They were what we call secondary renal calculi, and unless we get rid of the infection in those pelves, those stones will reform again and again.

We used to think infection of those pelves occurred by way of the ureter, but have now given that up. We know, on the contrary, that the kidney can excrete organisms from the circulation without any apparent damage to the organ. We know that tubercle bacilli in the urine do not prove tuberculosis of the genito-urinary tact. We know that the Bacillus coli is found in the urine of 1 in 20 women, and kidneys suffer no harm.

If, however, a kidney be weakened by trauma, the right kidney may be mobile unduly, or by pressure on the right ureter by a pregnant uterus the organisms can then produce definite lesions demonstrable clinically. I have said to you that in order to cure this type of secondary stone the infection must be cured.

Of course, infection does not play so great a part in renal calculi as it does in gall-bladder calculi. Schade has done pioneer work in the elucidation of the primary renal calculi; the stones unassociated with infection through this way occur later. To follow Schade's explanation of primary stone formation we must comprehend what he means by the terms "colloids," "reversible" and "adsorption," as applied to this problem. Colloids are substances which appear to dissolve in water (*e. g.*, mucin or fibrinogen), but are probably in a condition of suspension, since the osmotic pressure is not affected by their presence. A reversible substance is one which can be redissolved after being deposited from solution, for example, a crystal of magnesium sulphate. Mucin is a reversible colloid. Fibrin is an irreversible colloid. *Adsorption* is the property colloids have of holding in a solution inorganic substances to an extent greater than could be dissolved in an equal quantity of water. He would suggest that, for example, uric acid in urine is held there in its excess by adsorption, on urochrome and other reversible colloids.

Should the uric acid be in excess of the amount which the colloid can hold by adsorption, we can picture the uric acid being deposited on the colloid. Should the colloid be a reversible one, both colloid and deposit can be removed by more water. If the colloid be irreversible, *e. g.*, if that uric acid be deposited on the surface of fibrin, then no amount of water will redissolve that irreversible colloid—the patient is forming a primary renal calculus which no mineral water or drug ever cured. The colloid is the source of the small amount of organic matter found in every primary aseptic calculus. The tendency of fibrin to form laminæ is the reason of the laminations of these primary renal calculi, and, lastly, to cure that patient we have not only to diet him in future to prevent excess of the substance which formed the stone; we have not only to tell him to prevent relative excess by drinking abundance of water, but we have to discover the cause of fibrinuria, or fibrin in the urine, which occurs apart from hemorrhage. This problem has still to be solved.

Before concluding I must apologize for my voice. On getting up this morning I found myself very hoarse, and while talking have been picturing myself getting gastric ulcer, renal calculi, and everything else from the infection. Last Saturday I got the first number of this year's British Medical Journal. On opening it, the first thing I saw was the name of Dr. John B. Murphy. The article was written by Sir Berkeley Moynihan, whom we respect in England as one of the most brilliant men which it has been the good fortune of England to possess. It was an article on gall-bladder disease, and the author quotes Dr. Murphy and refers to him as one of the greatest surgeons that America has ever produced. I can only say, in my own humble way, as I have watched Dr. Murphy at work, that we all heartily agree with the writer, but it gives it much more force coming from such a man as Sir Berkeley Moynihan.

[Applause.]

Dr. Murphy: Aside from the last statement of the speaker, the talk was mighty instructive and mighty interesting, and just what we expected to hear from him. The talk has been a most interesting one to me, and I am sure as interesting to you. To use that expression I made on opening, and the observation that I made in London, we are fortunate in having with us one of the lights in modern surgery, and one of the men whose work will be recognized over all the world by just that clean-cut presentation of facts. Now, then, he has been very good in giving us such a splendid talk. We will ask him when we are doing the next operation to talk on any subject he may desire. His talk on ulcer made it very interesting to me in connection with this case.

FURTHER REMARKS BY MR. ROBERT MILNE, F.R.C.S., OF LONDON, ENGLAND

At Dr. Murphy's Clinic, following an operation by Dr. Murphy for fracture of the humerus and Colles' fracture

Dr. Murphy: While I am finishing this case I will ask Mr. Milne to talk to you again.

Mr. Milne: *Dr. Murphy and Gentlemen:* I was very much interested in that case as a Colles' fracture, but more especially in connection with the fracture of the humerus. It showed that Americans and English are alike human in that they both make mistakes. Although the median nerve runs a very close second, there is no nerve injury so frequently missed as that of the musculospiral.

This latter nerve is injured in one-tenth of the fractures in the lower half of the humerus. It supplies the skin on the back of the forearm, the extensor muscles of the fingers, and the extensors of the thumb.

The skin is supplied by branches which come off in the upper part of the arm, and therefore escape injury when the lesion is in the lower half of the humerus. Extension of the fingers is performed by the forearm extensors and by the interossei and lumbricals inserted into the extensor tendons. Since the lumbricals arise from the long flexor tendons, these latter are rendered tense when the lumbricals are in action. Therefore extension of the fingers involves contraction of the long extensors, the intrinsic muscles of the hand, and the long flexors. Combined action of muscles such as this is called "synergic action."

The point is this: a patient with complete division of the musculospiral can still extend his fingers at the interphalangeal joints by the interossei and lumbrical muscles supplied by the median and ulnar nerves, and he can still extend his thumb, because the abductor pollicis, supplied by the median nerve, constantly sends a slip to the extensor of the thumb, though, of course, one does not see in the latter case the tendons standing out and forming the "snuff-box."

It comes to this. When a man has his musculospiral nerve completely divided in the lower half of the arm, there is no loss of sensation, as he can still extend his thumb and he can extend his fingers at the interphalangeal joints. He cannot extend his wrist or the metacarpophalangeal joints of his fingers. It is no wonder these musculospiral injuries are missed frequently at the first examination, unless looked for especially. About a year ago a lad under my care illustrated this. The house-surgeon reported that this nerve could not be divided completely, as the boy could extend his fingers. On exploration I found the nerve lying between the raw surfaces of an oblique fracture of the humerus, very badly bruised, but it recovered its function, after being freed and wrapped in Cargile membrane, in ten days. I believe that in this case immediate operation on the fracture was justifiable, because if we had waited, the nerve would inevitably have been very seriously damaged by manipulation or in scar or callous tissue.

I should like to show you a band which I use for this type of oblique fracture. (*Vide* Fig. 40, *A* and *B*.) It is made of German silver, and can be tightened by screwing up the small square nut on the loop formed at one end of the band (*vide* Fig. 40, *A*), the thread being turned on the whole length of the band. As the nut is screwed up the circle of the band becomes smaller, and will encircle an oblique fracture more firmly than any wire that can be applied manually. The essential in applying such a band is the "spanner." The spanner, as shown in Fig. 40, *D*, surrounds the end of the band as it slips down on the nut. The second portion (Fig. 40, *E*), with its spiral spring outside and a screw-thread turned in its lumen, is passed along the interior of the "spanner," and by a turn or two grasps the threads of the band-

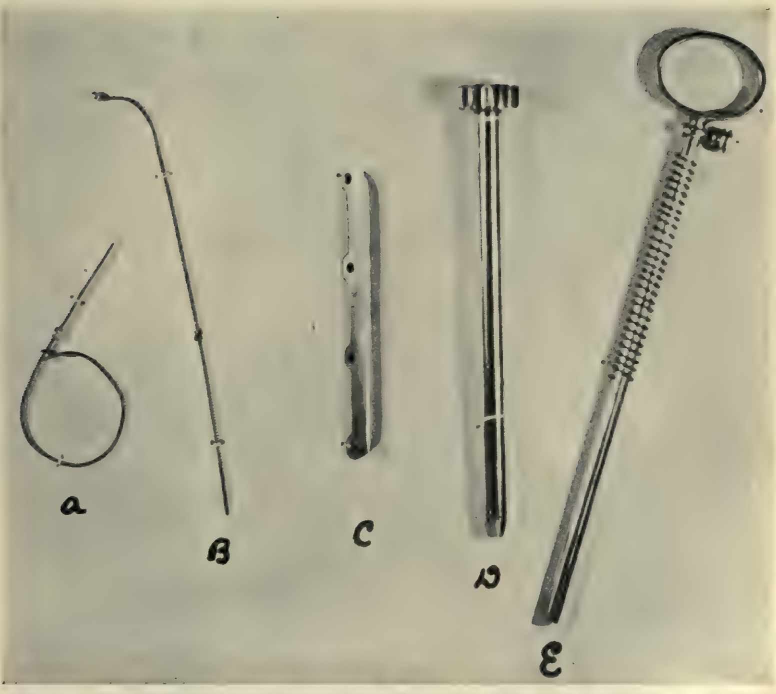


Fig. 40.—Mr. Milne's instruments for plating fractures. (See text for explanation.)

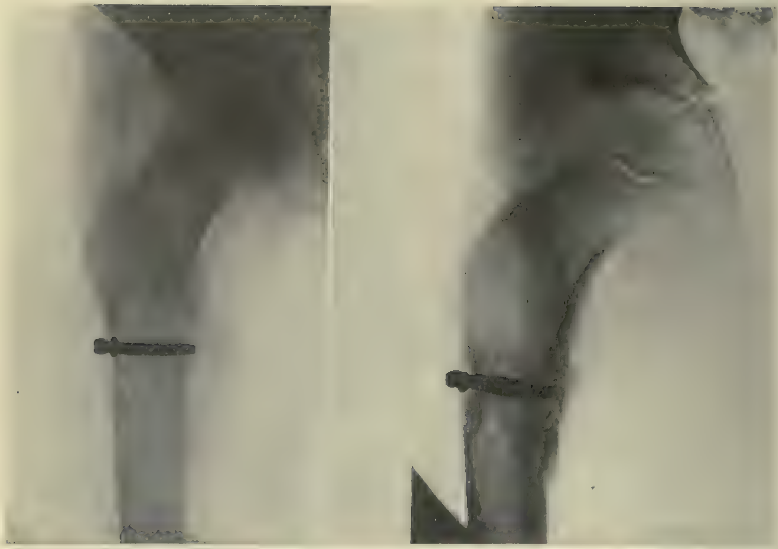


Fig. 41.—Fracture of shaft of femur wired by Mr. Milne. (See text.)

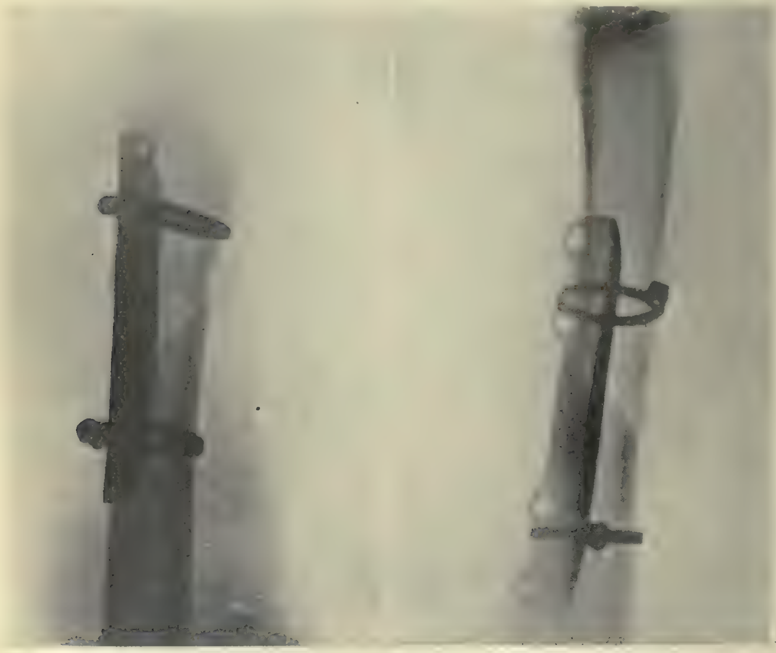


Fig. 42.—Fracture of femur plated by Mr. Milne. (See text.)

end. The spring now keeps a steady pull on the band inside the first portion of the "spanner," and presses this latter firmly on the nut as it rests on its upper expanded end. My description is bad, but the weapons are simple. The spanner and its spring are essential, for without the pull on the band the latter has a tendency to wander round the bone as the nut is tightened.

The band is cut off flush with the nut after application.

The objection might be raised that if you put such a band around the bones, pure pressure atrophy would lead to fracture later. In answer, look at Fig. 41. The fracture in the femur on the left was not very oblique, but it was the first time the band was used, and is, therefore, the oldest radiograph I have. The right-hand picture shows the condition eight months later, the band being buried on the inner side, the nut on the outer side being in process of burial. The whole weapon is easily made. This one was made in our London Hospital workshop.

I should like also to draw your attention to a new form of girder-plate, which was first suggested by Mr. Souttar, one of our surgical registrars, and is shown in Fig. 40, *C*. You know that a thin, flat piece of metal is not very strong. It we take that same piece of steel, say, bend it over longitudinally at an angle slightly less than a right angle, then we have added to its strength enormously, although we have still only a small amount of metal. That is the principle of this plate.

In a transverse fracture I get the ends of the bone in apposition, cut a slot along the upper and lower fragments across the line of fracture with a circular motor saw, and quickly drop into the slot the turned-over flange of the plate. The other flange rests on the outer surface of the fragments, may be held in position by machine screws, or, as I prefer, may be held by a band above and below. (*Vide* Fig. 42.) The bands are quicker, and, therefore, there is some less risk of an infected wound.

I should add that I operate on no fracture of long bones unless there is some positive indication, *e. g.*, nerve injury or failure of extension, splints, etc., and should like to insist on the credit due to Mr. Lane for impressing upon us the urgent necessity for absolute asepsis in these cases.

Dr. Murphy has told us of the necessity of an anesthetic for the reduction of fractures. We do the same in England, but we have a difficult class of cases in men who are addicted to alcohol. Recently it occurred to two of our men, Dr. Noel and Mr. Souttar, again, that the safest drug for these cases being paraldehyd, this might be used intravenously in a way similar to the use of hedonal or chloral, which are all, however, depressant drugs. You will find the method in the Lancet for September, 1912, but, briefly, they induced anesthesia with 5-15 c.c. of paraldehyd with an equal amount of ether, the mixture being dissolved in 150 c.c. of a cold 1 per cent. saline solution in sterile distilled water free from dead bacteria. Of the cases they record there, the major ones were mine, and I can answer for it that the effects were almost uncanny in their complete success. Every patient was completely unconscious in less than forty seconds, and there were no unpleasant after-effects. It was a most splendid and useful anesthetic.

[Applause.]

DR. MURPHY: How soon does analgesia follow the injection?

MR. MILNE: Forty seconds. The patients are absolutely asleep and ready for an abdominal operation.

DR. MURPHY: How long does it act?

MR. MILNE: The paraldehyd alone will keep a patient unconscious for from seven to ten minutes. We follow the paraldehyd with ether intravenously by an apparatus which kept the ether solution under pressure, and so allowed the saline to be warmed to body temperature, without the ether being driven off. I refused to allow ether to be administered intravenously to my patients in saline below body temperature. With this paraldehyd-ether sequence we have kept the patient under anesthesia for one and one-half hours.

DR. MURPHY: You get the regular ether analgesia as they use it in Heidelberg. We are again indebted to Mr. Milne for his instructive talk on the fractures and on the anesthesia.

CONTRACTION OF INTESTINAL ANASTOMOTIC OPENING WITH EXTENSIVE ABDOMINAL ADHESIONS; CECAL FISTULA

DISCUSSION OF MEDICAL PHASE BY DR. MIX

HISTORY

The patient, a male, forty years of age, comes to the hospital on account of continuous abdominal distention without very much abdominal pain, though with a good deal of abdominal discomfort. He has a cecal fistula. Five or six years ago he went to the west coast of South America on a hunting expedition with six companions. They went from the coast into the interior, and there contracted some sort of illness, the nature of which was wholly unknown to them. In the village in which they were taken ill many of the natives had died of the affection. Five of the party died. The patient and one other succeeded in getting to the coast. On reaching the coast he had a high fever, which was preceded by a chill and followed by a severe diarrhea. His friend died a few days later. The patient went to Mexico, and from there to San Antonio. The diarrhea lasted for three months, and was diagnosed as typhus fever. During the third week of this fever he had pain in the right iliac region, accompanied by nausea, vomiting, and abdominal rigidity. He was operated on and the appendix removed. From this time on he was an invalid. His statement as to his subsequent condition is not wholly clear.

During the last four years he has had nine laparotomies, his abdomen being scarred up by the incisions. Most of the operations have been done for the relief of adhesions. Notwithstanding all these operations, his condition did not improve. He has consulted various physicians. When he entered the hospital, he was in a condition of expansive mental alertness. On his arrival he slept only two hours out of the twenty-four for several nights in succession. Now he is controlled by paraldehyd, and gets about four hours' sleep each night. While in the hospital he has gained 15 pounds in weight.

COMMENTS AND OPERATION

Dr. Murphy: I will ask Dr. Mix to discuss the medical phase of the history.

Dr. Mix: The history, as given by the intern, although incomplete, has in it many of the essential details. The patient says he had typhus fever, and his statement may be correct. He also says that he had appendicitis and that his appendix was removed. He also told me that a portion of the cecum was removed at that time, but it is not likely that this operation was performed. We have obtained from Dr. Charles Venable, of the Lee Surgical Hospital at San Antonio, Texas, a statement as to the findings at such of the operations as were done at that hospital. Under date of March 12, 1910, a letter written by Dr. Venable states that at the operation which he had just performed he found a very extensive membranous pericolitis involving the whole ascending colon. He said that adhesions had grown across the colon from the cecum and the hepatic flexure, and that one of these adhesions had so constricted the bowel that the passage of feces was greatly interfered with. These adhesions were removed, but fear was expressed that it might be necessary, on account of the formation of new adhesions, to perform a secondary operation. This became necessary a few months later. In the interim between his operation for these adhesions and the subsequent operation for the new adhesions the patient developed some bladder irritability, which was thought to be neurotic in origin and which readily yielded to treatment.

By the fall of 1911 the symptoms had reached such a stage of intensity that it was proposed to the patient that his entire colon be removed. To this he assented, but at the time of the operation the adhesions which were found here were so dense that it was thought inadvisable at the time to remove the large bowel.

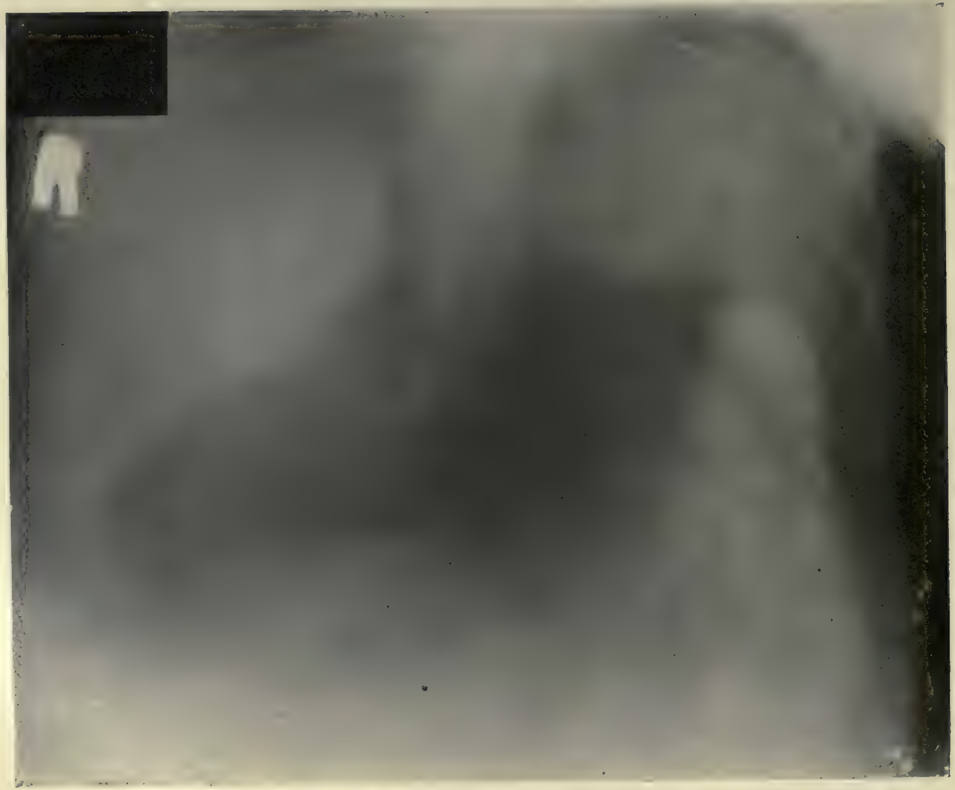


Fig. 43.—Abdominal adhesions. The proximal loop of ileum (light area on right) is "hung up" near the splenic flexure, where the intestinal anastomosis is made with the descending colon.

Dr. Venable then states that he divided the small intestine near the ileocecal valve, and that he attached the proximal end of the ileum to the sigmoid flexure on the left side. He did this because he found the constricted portion of the large intestine between the cecum and the sigmoid flexure. Within two days' time it became absolutely necessary, on account of the fact that no fecal matter passed from the bowel, to make an artificial opening into the cecum for the purpose of through-and-through irrigation. An opening was accordingly made in the right iliac fossa into the cecum. Into this artificial anus a tube was inserted through which irrigation was performed.

This operation was done some time in November, 1911, and for over a year the patient has been obliged to irrigate the colon twice daily in order to carry out the gas and fecal matter. The patient has observed that it takes nearly forty minutes for the fluid to pass from the artificial opening around to the rectum. He has also observed that the gas does not pass unless the colon is distended rather thoroughly. The patient gives his weight as 140 pounds, but owing to the great distention of the left side of the abdomen with gas, he looks as though he might weigh 170 or 180 pounds. Unlike an abdomen enlarged merely by fat, his abdomen shows the bulk of the enlargement to the left of the median line. During the patient's stay in the hospital he has had a good appetite, which he has satisfied, and by means of the irrigations has succeeded in keeping his alimentary canal in good condition. In fact, he has gained 15 pounds in weight. None of his stools are, however, formed, and he states that he has not passed a formed stool for over two years.

Feeling sure that there was an obstruction to the passage of his intestinal contents through the bowel, I had a series of skiagrams made. The first one of these was a picture of the stomach taken five minutes after the ingestion of 16 ounces of buttermilk containing 2 ounces of bismuth subcarbonate. A second picture was taken one and one-half hours, and a third two and one-half hours, after the ingestion of the bismuth. These pictures failed to show any definite block in the alimentary canal. Inasmuch as I believed that the block probably lay at the point of anastomosis between the small and the large intestine, I had him take the usual amount of bismuth and buttermilk by mouth at a certain time, and then two hours later caused an injection of bismuth and buttermilk to be made into the colon through his artificial anus. I anticipated that we might possibly find the junction of the two streams at the point of anastomosis. The expectations which we had were realized. The final skiagram shows a very thin stream of bismuth joining a very greatly inflated loop of the colon on the left side. The mass of bismuth in the small intestine as it apparently joins the large one in the sigmoid is rather attenuated, if the interpretation of this skiagram is correct, as we deem it to be. There is at the junction of the proximal portion of the separated small intestine with the colon a marked constriction which prevents the flow of the small-intestine contents into the large intestine. It would seem as though the patient, in his endeavors to clear out his small intestine, succeeds, by his irrigations, in distending the colon to such an extent that at the orifice of junction a sufficient dilatation takes place to permit some of the contents of the small intestine to pass into the sigmoid. Thus he intermittingly and imperfectly drains his small intestine.

The patient states that our conclusion is not in accordance with that of Dr. Venable, of the Lee Surgical Hospital, who, at his last operation stated that he found the opening between the small and large intestine sufficiently large for the patient's needs. We have advised the patient to have the abdomen reopened and to have this opening examined, and if, as we conjecture, it is too small, to have it enlarged. We also feel sure that there are other adhesions connected with the small intestine which interfere with peristalsis, and which, if possible, should be liberated.

Dr. Murphy: We will make the incision over the right rectus muscle, half an inch from the median line, and make it long enough so as to give us a good exposure. There is a large mass of adhesions here. The intestines are matted together and enormously dilated. The anastomosis between the ileum and the descending colon is contracted down to such a small diameter that considerable peristaltic action of the bowel was necessary to force its contents through the opening. The result is this hypertrophy of the bowel which you see and distention of the intestines. That also accounts for his intense abdominal distention. I am just feeling my way here, so that I will not get into trouble, meaning the bowel.

The large intestine below the anastomosis is not materially distended. The large bowel proximal to the anastomosis is not dilated. We will first carefully separate these adhesions in order to get a clear field. Then we can estimate the exact conditions present, and decide on our course of procedure. There are so many adhesions in the right iliac fossa that we hardly know where to begin the work. There is enough pathology here to more than account for all the symptoms manifested.

We are beginning to feel that our work will not be in vain. It appears that the ileum was divided close to the colon, and the end of the bowel was closed. About four inches from the proximal end of the ileum it was anastomosed laterally with the descending colon, just below the splenic flexure. The anastomotic opening has contracted down to almost the size of a lead-pencil. The small bowel is "hung up," as it were. The portion distal to the anastomosis is very much dilated. It looks like a Meckel's diverticulum. The catheter through which he irrigated his bowel passes down into the cecum. When the proximal end of the ileum was swung from the right to the left side, the adhesions on the right side that were freed before had become reëstablished, so that there was great tension between the point of anastomosis and the adhesions of the ileum in the right iliac fossa; further, the mesentery was not approximated to the posterior wall of the abdominal peritoneum to prevent the formation of an open loop. Through this open loop a large portion of the small intestine had passed, as we anticipated, and compressed the ileum, which passed across the pelvis from the right side of the small intestine to the large intestine, to which it was approximated. This spread out as a fan and produced retention by compression as well as retention by stenosis of the opening.

What this man needs is a sufficiently large opening between the small and the large bowel to permit the unobstructed flow of the intestinal contents and the entire freeing of the attachments of the ileum in the right iliac fossa so as to get this tension off. We will enlarge the opening now present, doing a typical suture operation. The large bowel does not need to be removed. The opening into the cecum will be permitted to close, because irrigations will no longer be needed if the flow of the intestinal contents is not impeded.

We have done all that can be done for this man, and feel that he will be benefited greatly by the operation. All of the small intestines were matted together in one mass of adhesions posterior to the compressing band of ileum, which could not be taken out and hole closed on account of the adhesions, and it was unnecessary because the hole was obliterated by the adherent intestine in it. The abdomen will be closed in the usual manner, suturing the peritoneum with catgut by making an ectropion of the cut edges; then the rectus aponeurosis, also with catgut. Silkwormgut figure-of-8 stitches through the skin and fascia and horsehair to approximate the skin edges complete the closure. We will put in a rubber tube drain because we fear that there may have been some infection of the cellular tissues with colon bacilli from the extensive and intensive handling of the intestines.

[This operation was of nearly three hours' duration, but the patient left the table in splendid condition. It was the longest laparotomy I have had in years. On the following day he had a normal bowel movement, the first in two years, and his bowels have continued to move naturally ever since. The tube was removed on the fifth day. The wound healed per primam. The fistulous opening into the cecum has almost closed. The abdominal distention has diminished, but the patient's condition is splendid. He is still gaining in weight. He had an accumulation of gas following the operation, which was rapidly relieved by alum enemas, two tablespoonfuls of alum to two quarts of water. This stimulated peristalsis.—ED.]

EXPLORATORY LAPAROTOMY, PERICHOLECYSTITIS; HEALED DUODENAL ULCER

HISTORY

Patient, a male aged twenty-eight, enters hospital on account of pain in the stomach and right hypochondrium.

Present trouble began in October, 1911, when patient spent ten days in the central part of Texas. On the ninth day he had a chill, which lasted one hour and was followed by fever. He does not know the degree of fever. Did not have another chill for three weeks, when he had four chills inside of sixteen days. They would last one hour, always came on at the same time of the day, and were followed by fever. He was taking quinin continuously. At this time he began to have an uncomfortable feeling in the right hypochondrium; sometimes a dull, aching pain which did not radiate. Stools were light in color. Urine was dark in color. He was not jaundiced. He was nauseated, but did not vomit. Belched a great deal of gas. The uncomfortable feeling in the right hypochondrium, with gastric symptoms and stool findings, has persisted.

One month ago began to grow weak and was compelled to give up his work. Has lost 10 pounds in the last year. Has had night-sweats off and on for six months. Bowels constipated; no cough. At present has an uncomfortable, heavy feeling after eating, coming on at once and lasting one to two hours. Never has a sharp pain. Nauseated at once after meals, never vomits. Belches a great deal of gas. Tender to touch over the stomach. Stools light in color. Has an uncomfortable feeling over most of the right hypochondrium. Heart negative. Lungs: Harsh breathing in the right upper lobe. Deep percussion gives a positive evidence of distinct ulcer under the central arch. No tumor can be outlined. He is somewhat anemic.

Was operated on by Dr. Murphy in June, 1898, for appendicitis. Malaria sixteen years ago, which lasted off and on for two years. Was living at the same place. Family history is negative. No tuberculosis. He smokes and chews to excess. Denies lues and gonorrhea.

COMMENTS AND OPERATION

Dr. Murphy: How long ago since he had the last attack?

Intern: Thirteen months ago.

Dr. Murphy: When I was a medical student malaria was very commonly mistaken for colic associated with gall-stones. Dr. Wm. E. Quine, one of the Nestors of internal medicine in this city, wrote a paper on Malarial Jaundice in 1892. He was the first in this country to recognize obstructions and infections in the common duct, with their associated chills and fever. In all the cases he described we now find stone in the common duct, with ulceration of the mucosa of the duct. That was the pioneer work in America. Trousseau had recognized the condition in France, and had written an account of it. It was believed that the chills and fever were an ague, although ague does not cause jaundice, while the real fact was that the chills and fever were the expression of the infection caused by the stone in the common duct. Since that time we have gained more accurate knowledge of this diversified condition. The gall-stones may be located in the biliary tract or in the common duct; there may be pain, associated with chills and fever, manifesting all the symptoms of malaria, but no jaundice. That occurs when the stone is outside of the common duct. When it is in the common duct, there is apt to be jaundice associated with the chills and fever. We have had many cases illustrative of this condition.

The patient must be asked whether he had chills and fever. Did the jaundice come on at a later period, and was it preceded by a colic. Gall-stone jaundice is always preceded by colic. This is a pathologic law. When a large stone passes down through the biliary tract, it never causes colic. It is the small stone which causes the colic. When the flow of bile into the intestine is obstructed, the patient has jaundice. Therefore an obstruction of the hepatic or common ducts is accompanied by jaundice. When the gall-bladder or cystic duct is obstructed, the patient does not have jaundice. The chills and fever are characteristic. They resemble the clinical picture of malaria very closely. The patient may have one or two chills in a day; he may have the four-day type, the three-day type, and yet no malarial infection, which Quine described.

In a case we had a short time ago the patient had had his chills and fever for eleven years. He had traveled all over the world to get rid of the chills and fever, without success. Gall-bladder drainage gave him immediate relief. We had another very striking example. A man had had chills and fever for seven years. His blood had been examined for plasmodia repeatedly, but none were found. Finally the surgeons came to the conclusion that he had a gall-stone infection. He was operated on and an infected gall-bladder was found. It contained a stone which had caused all his symptoms. That was eight years ago; he has never had another attack.

Still another man had chills and fever for eleven years. No stone was demonstrable at the time he was operated on. The gall-bladder was enlarged to the size of the second joint of my little finger. This was preceding the time when we were removing the gall-bladder as commonly as we do now. The first day of the attack which led to the operation he had a chill with a temperature of 106° F. He had two chills the day I saw him, and he had one chill the next day. He had been sick for fourteen days. We drained his gall-bladder and he has never had a chill since.

The temperature curve is very characteristic in these cases. The temperature rises quickly for an hour or two, and then as suddenly drops to normal. We have termed this the angle temperature, because of the abrupt rise and fall. It is easily differentiated from the temperature curve of typhoid. This man had this "angle" temperature.

What is the blood count? He says he was operated on for appendicitis.

INTERN: The white cells number 9000. Hemoglobin percentage is 80.

DR. MURPHY: We never open the abdomen in the midline. We go through the right rectus muscle in these cases. We divide the aponeurosis and retract or displace the rectus muscle to one side. We believe that this lessens the liability to hernia, because when the abdomen is closed, the muscle-fibers lie between the peritoneal and the skin incisions. The peritoneum is divided on the slant between two tissue forceps, so as to obviate the possibility of cutting the intestines which may be in the grasp of the forceps.

There is a cicatricial mass here in the gall-bladder region. It is the record of a former cholecystitis. How many years ago since he had his malaria?

INTERN: Fifteen years ago, doctor.

DR. MURPHY: I will palpate the gall-bladder and see if he has any stones in it. He has not. Let the record show that the gall-bladder is attached to the duodenum by adhesions resulting from a cholecystitis. There is evidence here that the infection endeavored to perforate the gall-bladder into the duodenum. The duodenum is bound to the gall-bladder, effectually preventing the infection from passing into the cellular tissues. The duodenum shows signs of a similar condition. It is difficult to determine whether the infection came through the gall-bladder into the duodenum or from the duodenum into the gall-bladder. The head of the pancreas is hard and indurated, showing that he has had an old infiltration. The pancreas is not fixed.

He has never had a colic. He never had a hematemesis. Quite a good deal of his pain was caused by these adhesions. There is no obstruction of the caput coli. The sigmoid is free. All his trouble was centered in the gall-bladder zone. The duodenum is angulated by these adhesions, which naturally obstructed the outflow of his gastric contents.

I have freed all these adhesions, and to prevent their recurrence I will tack the omentum in under and around the duodenum. A couple of tacking stitches of catgut will do that nicely.

Let the record show that there were evidences here of a small ulcer at the outlet of the stomach; that there was gastric retention, due to the cicatricial formation between the gall-bladder and the duodenum; that a portion of the stomach was attached to the gall-bladder. Let the record show further that he had an old cholecystitis which had attempted to perforate into his duodenum.

I make the usual closure of the divided peritoneum by turning the cut edge out. That will prevent the formation of intra-abdominal adhesions. The muscle is not sutured. It falls back into place as soon as the retractors are withdrawn and overlaps the line of suture of the peritoneum. The incision in the aponeurosis is closed with catgut, and then we put in a figure-of-8 stitch, close all the layers of the wound above the aponeurosis, and obliterate all dead spaces. We close the skin with horsehair. We use an inabsorbable suture. Horsehair is better than silkworm-gut because it is inelastic and does not cut transversely. The elasticity of silkworm-gut makes it injurious to the skin. The silkworm-gut also has a capillarity that can carry infections in from the skin along the line of suture. The horsehair has no capillarity. It may safely be left in the skin almost indefinitely. We seal the wound with collodion gauze. That is a routine procedure in this clinic. The usual dressing is then applied.

[The patient made an uneventful recovery and was entirely free from symptoms when last heard from. In three months he gained 27 pounds in weight.—Ed.]

DUODENAL ULCER; PERIDUODENITIS; GASTRIC ULCER WITH ADHESIONS; PERICHOLECYSTITIS; GALL-STONES

MEDICAL ASPECTS OF CASE BY DR. CHARLES L. MIX

HISTORY

PATIENT, a female, aged sixty-seven, enters hospital on account of distressing pain in the right hypochondriac and epigastric regions.

Present trouble began about ten years ago, when she complained of a sense of fullness in the epigastrium about two hours after eating. This distress was more marked after eating a full meal, and was not accompanied by nausea or vomiting. These attacks would last about two hours, and occurred every three or four weeks or oftener, if a large quantity of food was taken. Accompanying the distress the patient complained of abdominal distention and belched quantities of gas. This condition continued, occurring at varying intervals, until two years ago, when the distress became more pronounced and recurred at shorter intervals. She now complained of a gnawing sensation in her epigastrium, accompanied by a feeling of distress and belching of gas. This would invariably begin about two hours after eating, and continue for about two hours. Occasionally the distress would be located in the right hypochondrium, was never a distinct, sharp pain, but a feeling of pressure and fullness. These attacks would be accompanied by nausea, but never by vomiting. Attacks now began to come on every ten to fourteen days, and lasted three to four hours. For the past year patient has complained of a sense of fullness in the right hypochondrium and epigastrium, accompanied by belching of gas after nearly every meal, but principally the evening meal.

About three weeks ago the distress became more marked, and was present in the epigastrium and right hypochondrium after every meal. The belching became more distressing, and she had to sit up in bed to relieve the abdominal distress by belching. For the past two weeks the distress was so marked that she took morphin, and about one-half hour after taking it she vomited and continued to vomit all day great quantities of greenish-black, bitter, and offensive fluid. Following the vomiting the distress disappeared. Two weeks ago she again took morphin to relieve the distress, and again vomited quantities of blackish-yellow fluid and also blood. She has taken morphin five times, and has vomited one-half hour after each injection. Two weeks ago she says she was jaundiced, and she also noticed a rash over her entire body. This gradually disappeared in about ten days, and the skin became dry and scaly. Last attack of distress was five days ago, at 2 P. M., following an enema. Immediately after the enema she had pain in the right hypochondrium, extending well out to the epigastric region. This was followed by a feeling of distention, and belching followed. Has never had chills or fever. Says she has been jaundiced for the past two weeks. Is very constipated, and has been taking enemas for past three weeks. Thinks she has lost 20 pounds in weight in the last three weeks. Has had bearing-down pains in the pelvis for past eight years. Has to hold abdomen when she walks to give support.

Menstruation regular. Menopause at forty-three. Last period patient was sick for a month. Flowed for six or seven days. Noticed blood and pus in the urine. Had severe lumbar pain. Vomited several times and had a high fever.

COMMENTS AND OPERATION

DR. MURPHY: Dr. Mix, will you please discuss the medical side of this case?

DR. MIX: We have a history of preliminary attacks beginning ten years ago, associated with fullness in the epigastrium and epigastric pain, the pain radiating to some extent toward the right hypochondrium, being increased after a full meal and diminished by a small one. There is a further history of no nausea or vomiting, and, furthermore, to elaborate the history written by the intern, she often noticed that the taking of food would relieve the pain to some extent; for example, it occasionally happened that when she ate her supper, the pain which she had in the late afternoon would disappear. The points thus far accentuated are the facts that the onset of pain was two hours after eating; that it was occasionally relieved by the ingestion of food; that it was not associated with nausea or vomiting. There is another point of some importance in the history, namely, that the attacks came at intervals of three to four weeks, being separated by periods of normal health. The duration of these painful attacks was ordinarily two hours. During the continuance of the pain she complained of abdominal distention and the belching of gas. These symptoms are distinctly those of duodenal ulcer. The location of the pain, the time of the onset, the disappearance of the pain on the ingestion of food, the intermittence of the attacks with good health between, the associated abdominal distention and belching, the absence of nausea or vomiting, the presence of constipation, which existed, though it is not in the intern's history, point distinctly toward an old duodenal ulcer. Had we during this ten-year period been able to examine the patient's stomach, we doubtless would have found a hyperchlorhydria, and there might have been times when the feces would have shown the presence of blood.

According to the patient's statement, there was a distinct change for the worse two years ago, when the distress became much greater and the intervals of normal health became much shorter. The pain was distinctly epigastric, with much distress and belching of gas. Though it is not stated in the intern's history, the patient nevertheless told me that, beginning two years ago, the attacks of pain and distress began to come on about fifteen minutes after eating. She was never free from them unless the stomach was empty. We have no history that she vomited in them. She said she did not, so that there is consequently no history of vomiting of blood. During the last two years there is no evidence that she passed blood by the bowel.

There is a distinct epigastric soreness at the present time. These symptoms, taken in connection with the symptoms of ten years previously, lead to the inference of an ulcer of the stomach, the location of the ulcer probably being rather near the pylorus. From the history, the diagnosis of a duodenal ulcer beginning ten years ago and a gastric ulcer beginning two years ago seems indicated.

Another question arises: it is stated in the intern's history that during the last year there has been more or less distress in the right hypochondrium,—that is to say, in the region of the gall-bladder,—with a sense of pressure and fullness, and also nausea without vomiting. During the last year matters have steadily grown worse, so that three weeks ago the pain became so severe that she was given morphin. Altogether she was given morphin five times, and within one-half hour after its ingestion she vomited each time. The vomitus is described as greenish-black, but cross-examination failed to arouse suspicion that there was any blood in the vomitus, notwithstanding its color. When she vomited the pain was relieved. The last important fact is that she was jaundiced two weeks ago. This jaundice was not noticeable to the patient or her friends, but was observed by her physician in the conjunctivæ. At the present time the patient shows no jaundice whatever. She does, however, show a slight amount of exfoliating dermatitis, which came on about two weeks ago.

The last distinct attack which she had occurred five days ago, following an enema. In this attack the pain was for the most part located in the right hypochondrium and epigastrium.

As to the physical findings, the only noteworthy facts are the absence of any palpable tumor, of any pyloric tenderness, of any enlargement of the stomach, and of any stomach splashing. The Ewald test-meal showed a hyperacidity as to both free and combined acids. I gave her a motor meal, which I removed in four hours. It had all left the stomach except a few particles of food in the washing, which shows that she has no pyloric obstruction. After I washed out her stomach the pain disappeared. Within a half-hour it had recurred. When I washed out the stomach I found no evidence whatever of its enlargement. There was tenderness over the epigastrium, over the duodenal area, and over the right hypochondrium. Other than those, there were no physical findings.

Such a history points suspiciously toward gall-bladder disease—whether a cholecystitis or gall-stones or both remains to be determined. The absence of chills and fever does not aid very much in the differential diagnosis, because in many instances gall-stones occur without such chills or fever. At any rate, there is one thing certain: if gall-stones are present, there is no stone in the common duct or impacted in the neck of the gall-bladder. The jaundice which is said to have occurred must have been very slight, because no trace of it now remains. This absence of jaundice also indicates the absence of an impacted stone in either the hepatic or common duct. All the symptoms referable to the gall-bladder can best be explained on the basis of a pericholecystitis associated with, and probably secondary to, a duodenal ulcer beginning ten years ago.

The case in its entirety seems to have developed something like this: about ten years ago a duodenal ulcer appeared not far from the pylorus, which undoubtedly, in the course of time, produced a number of periduodenal adhesions, as such duodenal ulcers almost invariably do. Later—probably two years ago—another ulcer developed, this time in the stomach, the association of duodenal and gastric ulcer being by no means unusual. We feel sure that another ulcer developed because of the fact that she had a distinct exacerbation two years ago, and a condition of ulcer, because the pain, which formerly came on two hours after eating, now came on fifteen minutes after eating, and because of late there has been no diminution of the pain when she has taken food. It is likely that two years ago a gastric ulcer developed, that it has produced certain perigastric adhesions, which accounts for many of the epigastric symptoms.

Some time after the development of the ulcer—when, we cannot tell—the symptoms of gall-bladder disease began to assert themselves. This definite assurance which we have in the history places the development of the epigastric and hypochondriac pain rather earlier than the development of the gastric ulcer. A duodenal ulcer and periduodenitis often lead to pericholecystitis, and may, indeed, lead upward to the development of gall-stones. In this particular case we believe a pericholecystitis to be present, but we do not believe stones to be present, at least not in the common duct or in the hepatic duct, because of the absence of jaundice; because of the fact that vomiting relieved the attack; because of the absence of jaundice of a proportion sufficiently definite to be noted by the patient herself or her friends. Certainly there have never been any chills or fever or attacks of gall-stone colic. If stones are present, they must be quiescent. We would make a diagnosis, therefore, of duodenal ulcer; periduodenitis; gastric ulcer with adhesions; and pericholecystitis without stones.

Dr. Murphy: The conditions which make this a surgical case are: First, pain; second, food distress; third, gas. Did she have any food relief?

Dr. Mix: Yes, she had some, but it was not constant.

Dr. Murphy: We will open the abdomen to the right of the median line, going through the rectus muscle, between the umbilicus and the costal margin. She has adhesions between the gall-bladder and the stomach, which I am freeing. The stomach is pushed upward. There is some evidence of an old inflammatory process on the anterior stomach-wall, and there are some organic adhesions. She had an old ulcer, which tried to perforate through the wall of the stomach and fixed the stomach to the anterior abdominal wall.

Dr. Mix: That is why she had pain ten to fifteen minutes after eating.

Dr. Murphy: I must remove these adhesions so as to get that stomach free. This is the first case of such extensive and firm adhesions to the anterior abdominal wall I have seen. I will ligate and cut the adhesions. Now the stomach is free, and goes right back to its normal position. Those adhesions kept the stomach out of plumb all the time. They kept tension on it, and held it in a rotated position all the time. Now the stomach is free and movable in every direction. The stomach was roped by the band of adhesions on the lesser curvature, and these adhesions had drawn the lesser curvature up almost to the splenic flexure. The adhesions were firm. They looked like a white, fibrous band. I could lift the patient off the table by hooking my finger under that band. There is no hour-glass contraction of the stomach. There was a little contraction, due to the fixation. Four and one-half inches of the pylorus are free.

Now we will go on to the duodenum. By passing my fingers over the duodenum, you can see a portion of it slip out from under my fingers. There is the typical stellate scar on the anterior wall of the duodenum, the result of the healed ulcer. Adhesions have formed. We will free these.

Next, we will see whether she has developed gall-stones as a result of this extensive infection of the portal zone. There is one remark made by Dr. Mix to which I would like to take exception, namely, that patients with gall-stones are not relieved by vomiting. I am sure that he had reference to a condition where the stone is embedded in the cystic duct. Whenever the stones are embedded in the neck of the gall-bladder, the patient is relieved by vomiting, and she may have a hundred attacks of pain and vomiting before she will have jaundice. We will examine the pancreas. It is just a bit hard, not infiltrated, but has a greater resistance to touch than normal. There is no stone in the common duct and none in the cystic duct. She has stones in the gall-bladder. In fact, the gall-bladder is full of stones. It is small, white, and very thin-walled. It is stretched over these stones like a piece of parchment. That is why she did not have any gall-stone colic. We will enucleate this gall-bladder partially because there is no reason why it should be saved, and every reason why it should be removed. These stones never could pass out from the gall-bladder. The cystic duct is obliterated. The common duct is patulous. We will excise the gall-bladder below this mass of stones, and then turn in the cut edges of the peritoneum with plain fine catgut sutures. We will put a drain down to the stump into the hepaticoduodenal fossa.

I think that in these cases eructation of gas is a far more important symptom than nausea. We invert the cut edge of the gall-bladder, just as we do in a cholecystostomy for drainage, putting in the puckering-string suture, or invagination stitch above, and a second row of stitches below. Now we make the division of the gall-bladder. These stones looked like monoliths at first, but they are not. They are just particles of calculi that became impacted. There was a large stone in advance which prevented the smaller ones from going through into the cystic duct. I am digging out that mucosa of the gall-bladder stump with a curet and scissors, because it has no special work to do any more. The cystic duct has a narrowed, constricted lumen, and its walls are infiltrated. I am down to the neck of the gall-bladder. I will tie that portion with a ligature and put in a rubber drainage-tube outside of the stump into the connective tissue. I will not endeavor to put a drain into the neck of the gall-bladder itself. It is not necessary. I removed all the mucosa of the gall-bladder, leaving only the connective-tissue wall. It is just like the stump of the appendix. I am neutralizing that tissue with 95 per cent. phenol, to guard against infection.

VISITING DOCTOR: Do you use any alcohol after the phenol to neutralize it?

DR. MURPHY: No, because we want the phenol to continue its destructive action. I will cover that abrasion that would be left on the inner side of the liver to form adhesions with omentum.

Let the record show that the gall-bladder contained two masses of gall-stones fixed in an hour-glass contraction. The anterior stone was the largest, and blocked the opening into the cystic duct, so that the smaller stones could not pass out of the gall-bladder. There were adhesions, a pericholecystitis fixing the gall-bladder to the duodenum. There was also a fixation of the lesser curvature of the stomach to the anterior abdominal wall, about three inches to the left of the median line. This adhesion rotated the stomach on itself and caused considerable obstruction. The gall-bladder was enucleated down to the cervix and the end embedded and closed. A drain was passed down into the fossa and fixed to the embedded portion to take care of any possible subsequent leakage. There was a fixation and angulation of the duodenum, with an induration behind, near the head of the pancreas, showing where there had been a previous ulcer. The fixation to the anterior abdominal wall was the sequence of an ancient gastric ulcer whose symptoms were not clear.

The peritoneum, rectus aponeurosis, and skin are closed in the usual manner.

[This patient died of a double pneumonia on the fifth day.—Ed.]

EXHIBITION OF CASE OF TRAUMATIC BRACHIAL PARALYSIS

Operation Performed Two Years Ago

HISTORY AND COMMENTS

Patient, a male, aged twenty-eight, came to the hospital December 17, 1910 (two years ago), on account of inability to raise right arm. On November 3, 1910, he was shot with a revolver, the bullet entering at about the center of the right supraclavicular space, and remaining buried in the tissues. The man who shot him was about four feet from him, and directly in front of him at the time of the shooting, but the patient was rotated slightly to the left. Immediately after the shooting the patient's right arm dropped to his side and his shoulder fell. He was unable to raise the right arm to a right angle with the body, or to fold the forearm on the arm. He had lost the ability to pronate and supinate the forearm or extend the hand on the forearm. He was also incapable of extending the fingers, but could use all the flexor muscles of the forearm and hand. There were no sensory disturbances.

It was evident, as the result of our examination, that the bullet had passed through the lower portion of the brachial plexus, between the fifth and sixth cervical segments, and that it had divided the seventh and eighth cervical segments. The ulnar portion of the eighth cervical was intact, and the suprascapular nerve was functionating. The musculocutaneous nerve was partially intact; the greater portion of it was not. The median was also partially intact. The ulnar was completely intact. The musculospiral was entirely out of commission. The plexus was exposed by making a double division of the clavicle and reflecting the flap inward, and it was found that the cut nerveendings were in close approximation, without any tissue intervening, and, therefore, we believed that regeneration of the divided axones would take place. We approximated the cut ends very carefully, suturing them with fine catgut. Nothing else needed to be done.

September 27, 1911, he could use all the muscles except the long extensors of the fingers and thumb.

December 24, 1912, he has a full and complete extension of the fingers and thumb. There has been a complete regeneration of the divided nerves. All of his arm muscles are normally active. He has great strength in his arm, and a powerful grip. This case establishes the definite principle that regeneration of nerve axons can and will take place, with full restoration of function, if the approximation of the cut ends is done right. The ends of the divided axons must be in absolute contact, and remain in contact until the regeneration can take place. One must not be discouraged at the length of time which necessarily must elapse before there is a return of function. In this case two years have gone by since the operation was done. The result speaks for itself.

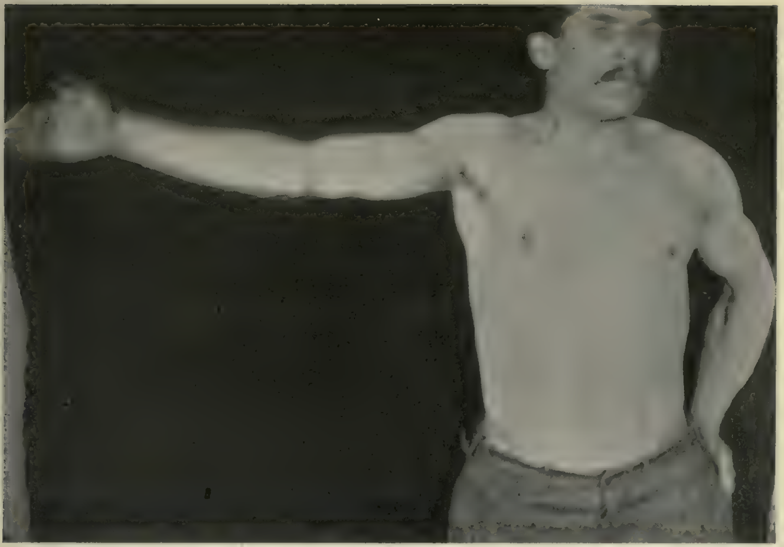


Fig. 44.—Showing results of operation done two years ago. Demonstration of case of traumatic brachial paralysis. Voluntary extension of right arm to right angle with body.

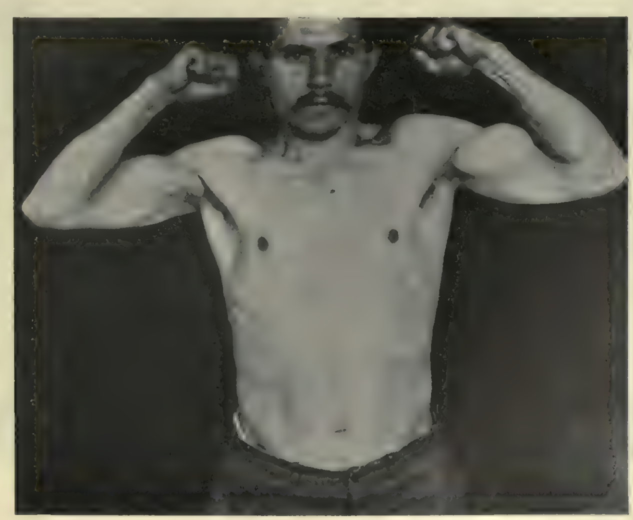


Fig. 45.—Showing muscular development in right arm equal to that of left arm.

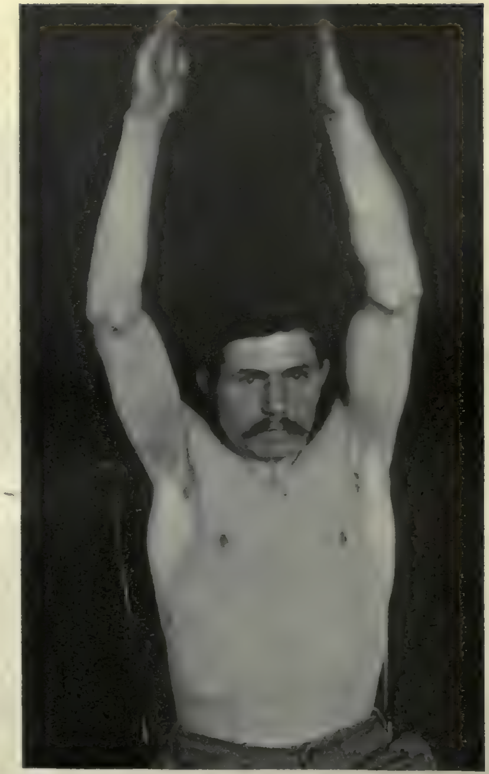


Fig. 46.—Both arms extended over head, with equal power in both.

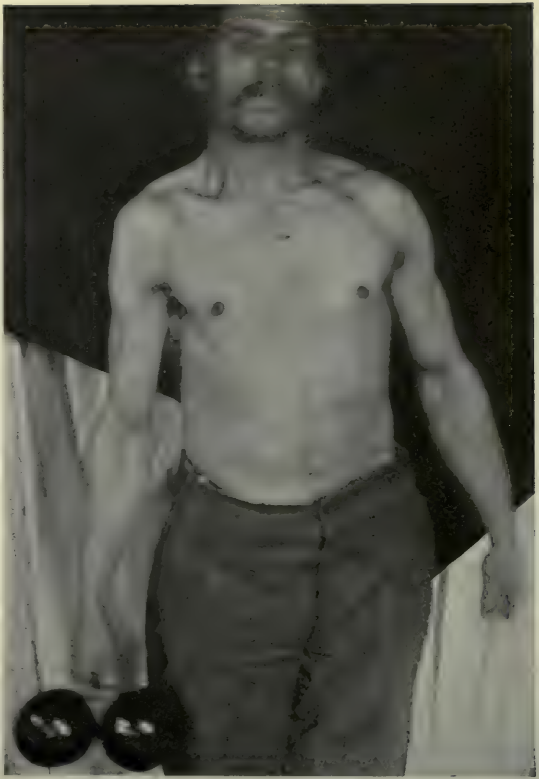


Fig. 47.—Holding a 25-pound weight in right hand.

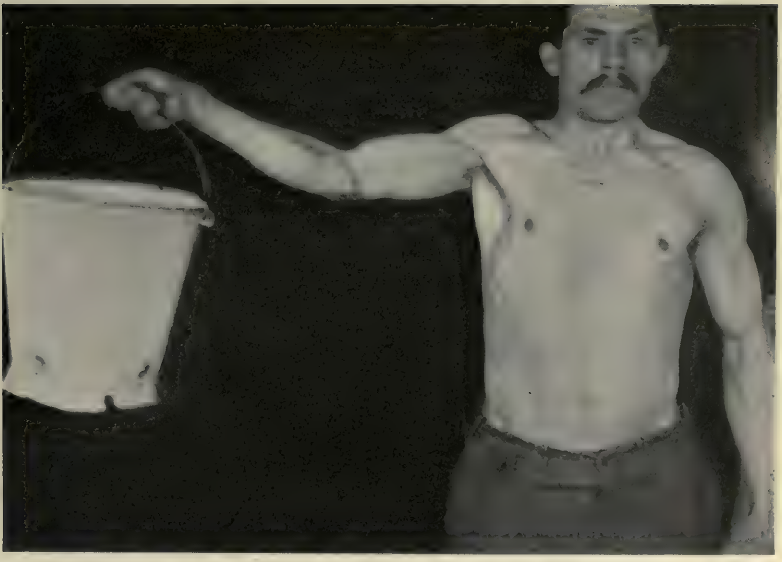


Fig. 48.—Holding a bucket full of water—about four gallons—with arm in full abduction and extension.

SPINA BIFIDA; MENINGOCELE

HISTORY

Patient, a male, one month old, was brought to hospital on account of a large mass over the spinal column in the lumbar region. Swelling dates from time of birth, when the attending physician noticed a sac about the size of a dollar. Five hours after birth it began to increase in size. It is now about as large as a hen's egg.

Normal delivery; no instruments used. Parents noticed fluctuations in size, especially when child cried or coughed. Consistence of mass varies from a soft, fluctuating swelling to a firm, tense mass. Parents also noticed a change in the color of the covering. Sometimes it is blue in color, but usually it is red. Child has retained motor function in both legs, and sensation is present. There is now an area of necrosis about 1½ inches in diameter, which started seven days ago. Baby has lost some weight.

COMMENTS AND OPERATION

Dr. Murphy: That is the usual story of this class of cases.

Spina bifida is a congenital malformation of the spinal column, meninges, cord proper, or ependyma, consisting of a protrusion, either of the meninges or of meninges and spinal cord, through a cleft, resulting from failure of coalescence of the borders of the neural groove, or absence of the vertebral arches.

The nerve-elements of the spinal cord are of ectodermic origin exclusively. The cord is an invagination of the ectoderm, forming a groove which is widely open at first, gradually closing to form a tube—the neural tube. The mesoderm supplies the serous, bony, and muscular elements, which arrange themselves around the neural tube. A malunion or absence of the involuting mesodermic elements which form the vertebral arches and spinous processes will leave a cleft through which the meninges alone, or together with the spinal cord, will protrude posteriorly, because of lack of support.

The defective coalescence of the spinal processes may be either partial or complete. In the first instance the opening involves two or three vertebræ, either in the lumbar or cervical region, as these are the last to close. The lumbar is the more frequent. Complete absence of union is called "rachischisis totalis," and is an uninterrupted opening, extending from the cervical to the sacral regions. This variety, however, is extremely rare.

In the circumscribed type of spina bifida we find a protruding tumor, the size of which may vary from 3 to 15 cm. in diameter, having either a broad or a pedunculated base. The sac usually protrudes posteriorly. Occasionally—very seldom, however—the sac bulges anteriorly; it escapes through the defective body or process, or through the spinal foramina, as shown in the following cases, and is discovered by accident. Several cases of this type are recorded in the literature. De Forest Willard reports a case—a child two months old, with a large cyst palpable through the right iliac fossa. The cystic tumor, which communicated directly with the spinal canal, bulged anteriorly when coughing. Its walls were the meninges, and the contents, cerebrospinal fluid. Emmet reported a case—a woman, thirty-six years of age, with a very large cyst extending down into the pelvis. It was aspirated through the rectum, and three quarts of fluid extracted. The patient died on the seventh day. Postmortem examination revealed a cyst formed of meningeal membranes, which protruded into the pelvis through the sacral foramina. Robinson operated on a case, a child eleven months old, who had a large cystic tumor occupying the entire right side of the abdomen. The patient had club-feet; there was no tumor to be found posterior to the sacrum. A diagnosis of parovarian or broad-ligament cyst of fetal origin was made. During the operation the surgeon found that the cyst communicated directly with the spinal canal, and that it was an anterior

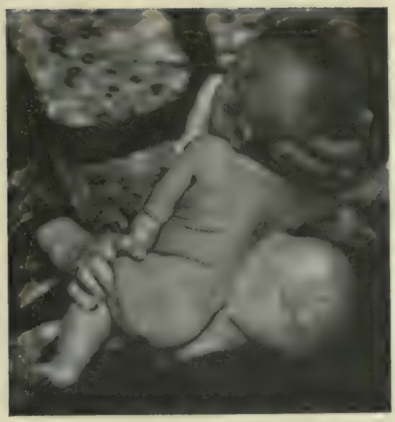


Fig. 49.—Spina bifida, showing large meningocele with beginning necrosis of the skin—the mottling over the top of the tumor.

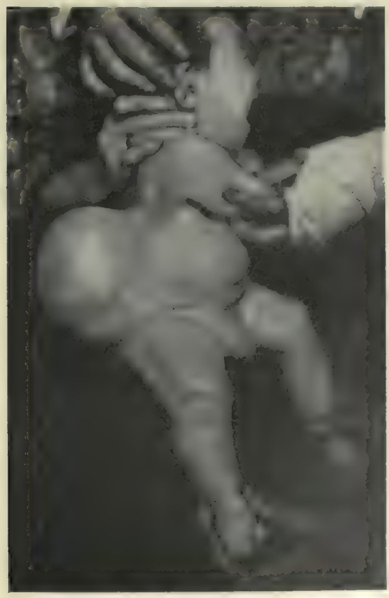


Fig. 50.—Spina bifida, showing another lateral view of the tumor from the right side.

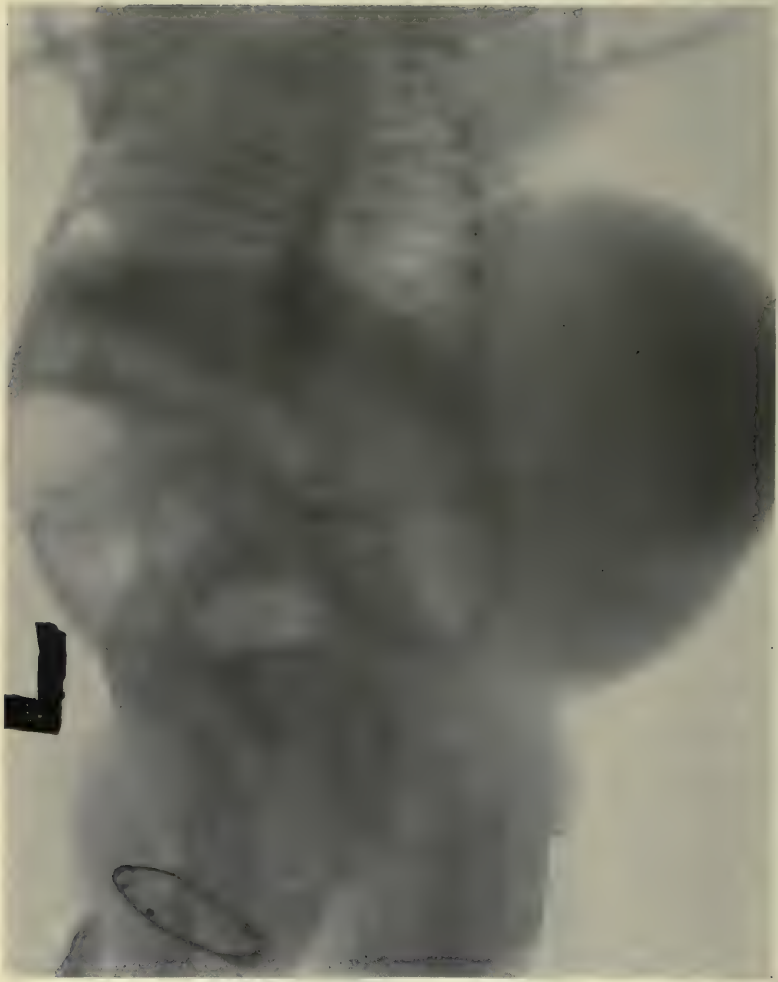


Fig. 51.—Spina bifida. In this skiagram the defect in the spinal column is evidenced at the base of the tumor shadow by the absence of definite dark shadows. The size of the meningocele in comparison with the body of the child is well shown.

spina bifida. Patient died after ten days. Postmortem showed extensive defects on the right side of the last dorsal and first lumbar vertebræ. The pedicles of the transverse process of these vertebræ were absent. The first two lumbar vertebræ had an undeveloped point of ossification on the right half, while the third lumbar vertebra had no point of ossification at all. The spinal column was curved laterally, the concavity being directed toward the left. There was a marked dilatation of the ependymal canal. Bryant reports a case of anterior spina bifida in a woman twenty-five years of age, who died from a trauma. The tumor was discovered accidentally.

Dana distinguishes three varieties of *spina bifida:*

1. Meningocele: A protruding sac composed of meningeal membranes and cerebrospinal fluid only. This may be either: (1) Anterior, abdominal, or pelvic; or (2) posterior or dorsal tumor.

2. Meningomyelocele; Hydromyelia: A tumor composed of meningeal membranes, cerebrospinal fluid, and spinal cord, including cauda.

3. Syringomyelocele: Composed of meninges, cerebrospinal fluid, and spinal cord, with an enormous dilatation of the central canal or ependyma.

To these we may add—

4. Spina bifida occulta, which is a cleft of the spinal column without any visible protrusion of the contents of the spinal canal. In great defects the cord is a velvet-like band containing blood-vessels and flattened nerve-elements spread out under the meningeal covering. In circumscribed varieties the sac may be covered with skin and hair, or simply with a thin epithelial layer, which, together with the pia, forms an epithelioserous covering.

In the first variety of Dana the cord elements may be intact. In the second the cerebrospinal fluid lies in the subarachnoid sac. The nerve-elements of the cord protrude into the sac in two-thirds of the cases. The fibers are attached on the posterior and median surface of the sac, *and form a part of the cyst-wall.*

In the third variety there is a considerable dilatation of the ependymal canal, with complete, or almost complete, pressure atrophy of the spinal cord at the point of dilatation, with a paraplegia below that zone.

If the dilatation had been two inches higher, operation for the cure of the paraplegia would have been worse than useless, and dangers of operation greatly increased, for in that position resection of sac and restoration of conformation of spine are all that could have been undertaken or justifiable. It must be borne in mind that the sacral and lumbar nerves pass upward a considerable distance in the spinal canal before they enter the cord or conus and have their neurilemma.

The etiology of spina bifida is very obscure. Occasionally it is one of a series of congenital malformations. A plausible explanation for the ill closure of the medullary groove is the traction by amniotic bands, preventing the union of its borders. Still, it appears to us that there is also an element of increased pressure from within, as in the anterior varieties failure of cleavage does not occur, but pressure seems to force the meninges through the foramina and bodies.

The general nourishment of the individual is usually below par. The mentality is defective. In cases of meningocele there are no motor or sensory disturbances. In meningomyelocele and syringomyelocele there are usually sensory disturbances, sphincteric derangement, and, not uncommonly, motor disturbances. In the latter variety paraplegia is usually present. Associated with the motor and sensory disturbances we find club-feet, spinal curvature, and other deformities.

If the spina bifida is posterior, the diagnosis is generally not difficult, except in spina bifida occulta. In this type the vertebral cleft is covered with skin and hair, and there is no protrusion of the cord or membranes. Patients affected with this variety, however, are not free from symptoms; we may find in them anesthesia, club-feet, and perforating trophic ulcers. The cases of anterior spina bifida are impossible of diagnosis before abdominal section.

Treatment consists either in injection or excision of the sac. Injection has long been and still is in vogue. Morton's fluid, which has the following composition, is the one generally used: Iodin, 10 grains; potassium iodid, 30 grains; glycerin, 1 ounce. Injection is not free from danger, and its efficacy is very questionable. We used it three times in a case without effect, except a temporary disturbance of the central nervous system.

Excision of the sac seems to be the radical and more desirable procedure. The sac is best attacked from the side, because the fibers of the cord are more frequently spread and attached to the median portion of the posterior wall, within the line of embryonal cleavage. When the sac is pedunculated, it should be first freed from its attachment to the skin and neighboring structures, except its highest point, which is adherent to the skin; there an elliptic cutaneous flap should be permitted to remain attached to the sac. When the latter is freed to a level with the cord, a provisional ligature should be thrown about its neck without tying. The sac should be opened, carefully examined, and if the cord is not protruding, the ligatures should be firmly tied; an overstitch of catgut should give it additional support. If the base is broad, the same procedure should be followed, except that after opening the sac and inspecting it the ligature should be removed. It should then be amputated at its base and carefully sutured if it be a case of syringomyelia, and if the cord protrudes into the sac, the fibers of the cord should be freed from their attachments to the meninges and replaced in their normal position, and the opening in the sac closed with a continuous chromicized catgut or, preferably, kangaroo tendon suture. If a complete syringomyelia exist, the cord should be amputated in both directions from the center of elevation to a normal anatomic zone; then a careful end-to-end suture of fibers should be made if the spina bifida be situated in the caudal zona. If above the caudal zone, the cord should be freed and replaced within the dural sac without suture, the opening closed, with no hope of motor improvement offered the patient. In localizing the muscles supplied by the distal fibers the faradic current should be used. Our results have been most satisfactory after this technic, both immediate and remote.

Mayo-Robson uses a median incision. He also leaves a segment of the skin in place if separation of the dura from it is difficult. He advises that the meninges and skin should not be united on the same longitudinal line. In the clinic of Breslau the meninges are united longitudinally and the skin transversely. After removal of the sac, in order to prevent subsequent hernia of the contents of the spinal canal, the defect is repaired either by a muscular flap (Bayer) or by osteoplastic flaps (Selenko Boboroff). Mayo-Robson once transplanted periosteum and bones from animals for this purpose.

Of 647 cases of spina bifida which died in England in 1882, 615 were under one year of age. Of the 90 not operated upon, the great majority died within the first few weeks. Only 20 reached more than five years of age. The mortality in the treatment of spina bifida by injection of Morton's solution is 20 per cent. Statistics of the Clinical Society in London, which include 71 cases treated by injection, showed 49 per cent. cured, 5.4 per cent. improved, 7 per cent. unchanged, and 38 per cent. mortality.

The following classification will serve to summarize the important phases of this subject:

VARIETIES:
I. Myelocele. (All fatal.)
II. Syringomyelocele. (Most of them fatal.)
III. Meningomyelocele. (Large percentage fatal.)
IV. Meningocele. (All recover.)
V. Masked (occulta). (Non-operative.)

COMPLICATIONS:
I. Paralysis of extremities.
II. Deformities of extremities.
III. Hydrocephalus.
IV. Intestinal occlusions.
V. Infection.

DEFECT IN BONY CANAL AND POSITION OF ESCAPE:
I. Between laminæ.
II. Through spinal foramina (also sacral).
III. Anterior or anterolateral, through absence of vertebra—body or transverse portion.
IV. Anterior, posterior, lateral, and combinations.

TREATMENT:
I. Palliative.
II. Surgical.
III. Injection.

This case, I think, is one of meningocele, not involving the cord, because all the muscles of the lower extremities apparently are active. There is considerable necrosis of the skin, which is always a serious complication in this class of cases. The baby is only four weeks old, and an operation of this extent is a serious one, but if we allow this condition to go on to the degree of ulceration,—and the necrosis is certain to become an ulcer,—the spinal fluid will be exposed to infection, and 100 per cent. of those infected cases terminate fatally. It is for that reason that we are going to do an operation involving so much hazard.

We will place the baby on the side with the head down and the body raised up, because in that way you lessen the loss of the cerebrospinal fluid which always escapes in this operation. It is important to preserve as much as possible of the cerebrospinal fluid. In some recent publications on the subject it is suggested that the child be suspended by the feet. Placing the child in this position also lessens the loss of blood, which is usually great in these cases. These patients are greatly hazarded by that loss of blood.

We must preserve a considerable portion of this sac in order to get sufficient skin to cover this area, and not have any tension on the sutures. We do not use any tincture of iodin here, merely alcohol and ether. The incision through the skin encircles the patch of necrosis, going about one-half inch beyond its margin to insure a healthy skin-edge. Then we carefully free the dura from its adhesions to the skin, freeing the sac from all its attachments and then excising it. There is the caudal zone. The meningocele is situated below the conus. There is the cord. There is some dilatation of the ependymal sac. We will remove the spinous processes of the last dorsal and first lumbar vertebræ with bone-cutting forceps. We know, from our work in laminectomy, that this spinous process is entirely unnecessary to the proper function of the cord, and that its absence does not weaken the strength of the spinal column.

Let the record show that this was a hydrops of the cord; a meningocele; a spina bifida involving only the meninges; that the cord itself was not involved in any way, so far as we could ascertain. The dural sac was first freed from the skin. There was a dense, fibrous, connective-tissue mass, showing the coalescence of the embryonal layers. This was then freed and the margins of the dura freely exposed. The sac was freed and excised. Accurate apposition of the dura, arachnoid, and pia was made with fine chromicized catgut. The muscles were sutured over the spinal column, so as to cover the defect. The skin-edges were carefully approximated with horsehair. There was no tension on the skin. Collodion gauze sealed the wound after it was well dusted with bismuth subiodid. A large dressing was then applied. Little spinal fluid escaped during the operation.

The child must be kept lying on its face and abdomen, so that the urine and feces will not infect the wound by saturating the dressings. There was much less blood lost than is usual under these circumstances.

[Everything went on very nicely with this child until the eighth day after the operation, when it had a severe convulsion and died. The cause of the convulsion was not ascertained. There was primary wound healing; motion in the extremities was perfect after operation.—Ed.]

IMPACTED FRACTURE OF THE BODY OF THE FIRST LUMBAR VERTEBRA; LAMINECTOMY; RAPID RECOVERY FOLLOWING DECOMPRESSION OF CORD

DISCUSSION OF NEUROLOGIC PHASE BY DR. PETER BASSOE

HISTORY

PATIENT, a male, aged thirty-five, comes to hospital on account of injury to spine in dorsolumbar region.

On the night of July 26, 1912, he walked out of his room to a porch in his sleep, and fell from the porch to the ground, a distance of about 12 feet, landing on his buttocks. He was unable to get up on account of pain in back. He was able to move his legs, but it hurt him to do so.

When put back to bed, his doctor found a prominence of the last dorsal and first lumbar vertebræ, with ecchymosis in this region. Examination then made showed no muscular paralysis, but loss of sensation over buttocks, perineum, scrotum, and back of thighs, as far as knee; no loss of sensation in front of thigh; no girdle pain and no annular paralysis. He felt for a few weeks as if a tight band was placed around each thigh.

Since the accident he has had to be catheterized thrice daily; could not pass any urine. For the past week or two control over bladder seems to be returning. With an effort he can pass a little urine now. For the first four or five days he had great trouble in getting his bowels to move; since then he has had involuntary evacuations. For the past week he is getting a little control over his rectal sphincter.

Examination reveals a prominence of the eleventh and twelfth dorsal and first lumbar spines. No paralysis of muscles of thighs, but calf muscles feel flabby. Tactile sensation absent over glutei muscles and diminished on back of thighs. No loss of sensation over abdomen. Superficial reflexes present. Left knee-jerk exaggerated. Right slow and scarcely perceptible.

Right ankle was swollen and somewhat painful after accident. Previous to accident patient was in excellent health. Heart and lungs normal. No luetic history. Family history negative.

COMMENTS AND OPERATION

Dr. Murphy: In other words, he has what? He has a saddle analgesia, which is so typical of injuries to the spinal cord in this zone. I am going to talk simply about the physical portion of the injury.

When you look at these two skiagrams you can see that the lesion is at the first lumbar vertebra. According to the first skiagram,—an anteroposterior view of the spinal column,—you would say that there should not be sufficient trouble here to interfere with function. But turn to the second skiagram—a lateral view of the spinal column—and that is usually the one that is made and the only one that is made; it shows that the anterior portion of the body of the vertebra is crushed—not only broken, but crushed, without luxation. The body of the vertebra forms a wedge, the apex of the wedge being directed forward. That throws the spinal column backward and the pressure on the cord is at that point. It was fortunate that when it was fractured it was not displaced, in which case it would have cut the cord at the conus. The conus comes to the upper margin of the second lumbar vertebra. The major portion of the lumbar cord is situated just at the upper margin of the first lumbar vertebra. It is one of the danger zones for trauma. In the center of the cord in that zone are the axons that supply the saddle zone of paralysis in this case.

This is a beautiful case for analysis from a nerve standpoint. This is a little lower than the usual break. The usual break is higher up—in the twelfth and eleventh dorsal vertebræ. When a man falls on his shoulders or buttocks, that is where he doubles and there is where the fracture occurs. He has a broken vertebra, one lower, at least, than the average break comes, and he has,

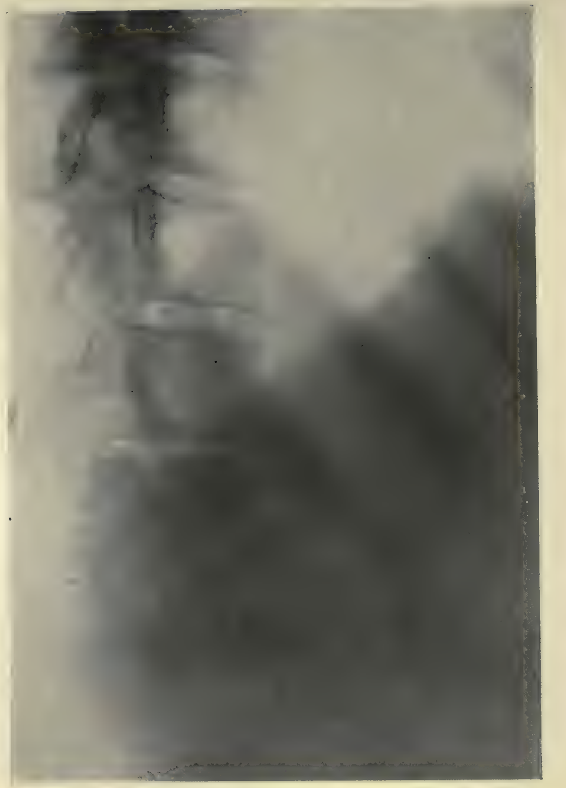


Fig. 52.—Case of laminectomy. Skiagram showing telescoping of anterior portion of body of first lumbar vertebra, with backward angulation of the spine. The skiagram shows that the body of the vertebra is wedge-shaped, the tip of the wedge being directed forward.

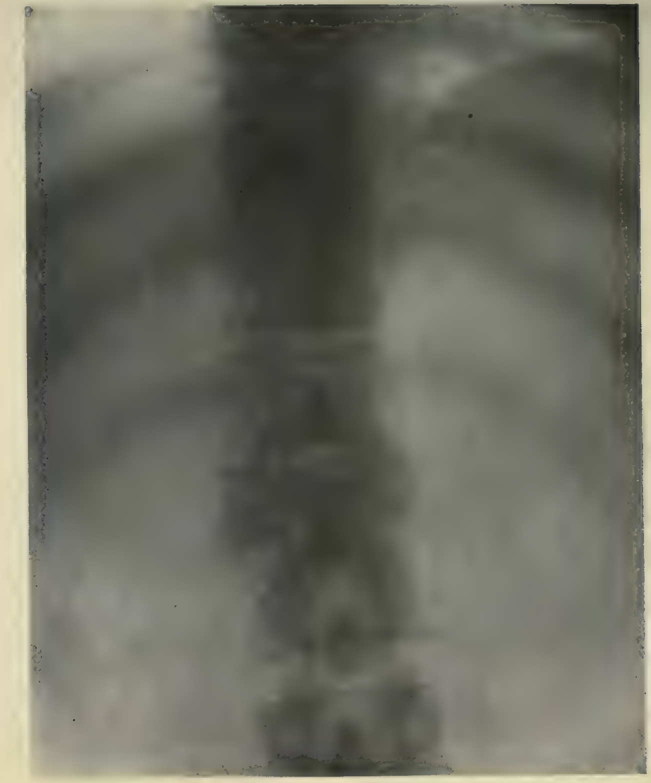


Fig. 53.—Case of laminectomy. Skiagram, postero-anterior view. The spinal column is straight and apparently normal. No pathology is evident. The lateral view showed a decided deformity.

therefore, the assurance of preservation of function of most of the spinal cord centers as well as their axons of his sacral or sciatic plexus, and all of his anterior crural or lumbar plexus, if the new osteal production does not encroach on the lumen of the spinal canal. The sciatic cord segment is situated above. If the medullary portion of the cord were cut in that trauma, he would never have a regeneration of the part that is now in anesthesia or analgesia, but it is my belief that he has suffered only a trauma or contusion of the cauda, and that his paralysis is the result of an edema of the cauda; in other words, there is only a suspension of transmission, and therefore function should be restored.

We are fortunate in having with us this morning the man who referred the case to us, Dr. Peter Bassoe, who knows more about nerves than I do, and I would like to have him explain to us the nerve phenomena as presented in this case and give us his opinion of it.

DR. PETER BASSOE: I do not believe that I can add anything to what Dr. Murphy has said about this case. When the patient came to me he gave the history you have just heard, and the chief trouble was his loss of control of the bladder and rectum and a loss of sexual power. On examination, as Dr. Murphy said, a typical picture of a conus lesion was found, and there was no difficulty at all in determining that we had to deal with a conus and a cauda equina lesion—a point of practical importance when it comes to the surgery of the cord. Of course, dealing with a fracture and having the skiagram, we did not make our diagnosis on the physical findings.

In a cauda equina lesion there is nearly always a good deal of paralysis, and the anesthesia is usually more extensive and goes farther down on the legs than it is when it involves the segments higher up. The anesthesia here involves only the region around the anus, the buttocks, the perineum, and the genital organs. Sometimes we also get the lesion involving the first and second segments, and, in addition, we get a paralysis in the area of the sciatic nerve and loss of ankle reflexes. The ankle reflexes in this case were not obtained by me; I do not know whether they were taken here at the hospital. The question that concerned me before I saw the skiagram was whether this man had a hemorrhage into the conus or whether there was an injury to the bone. Of course, the skiagram cleared all that up completely. I advised operation after seeing the skiagram, largely because I thought there is danger of a subsequent trauma increasing the deformity and causing a sudden paralysis, and also because I was afraid that a callus might form later on and compress the cord.

Dr. Murphy: The general management of this class of cases is a very important one, from a surgeon's standpoint. Some years ago I wrote an article on Surgery of the Spinal Cord and Peripheral Nerves, in which we laid down some general rules that we have found to be of definite value. Wherever you have an injury below the tip of the conus, that involves, or cuts, or compresses the cord, the line of action is clean cut for an immediate operation; no matter whether the nerves are cut or not, that is below the tip of the conus, and that must involve the body of the second, third, and fourth lumbar vertebræ. If they are compressed by a displaced bone, it will produce permanent paralysis unless the pressure is taken off. If they are cut and are reapproximated with a simple suture, the conus portion of the nerve is capable of regeneration, in exactly the same way as in the peripheral nerve, so that you have all of the indications for action that you would have if the nerve were compressed between bone, as the musculospiral sometimes is in fractures of the arm, or if it were cut by an ax or a knife, because you have in the cauda the conditions necessary for regeneration, namely, the axons have a neurilemma and they are therefore capable of regeneration.

It is entirely different, however, when you get into the conus zone, and when you go from the conus zone up through the spinal cord. If the spinal cord is cut in the conus or above the conus,—cut or completely divided,—any operation is contraindicated, because there is no regeneration of the axons that are within the true cord, because the axons in the cord have no neurilemma, and once they are divided, they are incapable of regeneration.

The difficulty of determining in the individual case whether the cord is simply contused and continually compressed and there is merely suspension of transmission, or whether it is cut and there is a complete division of the axons, or whether you have a reaction to the trauma in the way of edema and swelling, which have prevented transmission, we endeavored to bring out in our article, and we laid down some laws governing action. When paralysis of motion and sensation was on the same spinal center level, completely annular, and instantaneous following the injury, the cord in all probability was cut and an operative procedure should not be resorted to. When the paralysis is irregular; when it came on a few hours after the injury, then the cord was not cut: it was either compressed or it is a traumatic reaction to the contusion, and operation is indicated to relieve the compression, and the operation, if done at all, should be done immediately.

Now the question comes up, What are we to operate on this patient for? We are to operate on this patient not to cure the deformity of the body of his vertebra, but to remove a lamina. Why? Because by removing a lamina you relieve the compression on the cord. In all of the operations of this kind we remove the spinous process. It is the first lumbar vertebra that is involved. It is there that we wish to decompress the spinal cord. What do I mean by decompressing the spinal cord? I mean, give the spinal cord an opportunity to escape backward from any pressure that may come on it from in front; I mean to take off a lamina and the spinous process. See how that deformity stands out on this man's back. The transverse process is also palpable. That corresponds exactly with the skiagram.

We will make an incision in the dorsolumbar region directly over the spines of the vertebræ, so as to give us free access to the spinal column. You can all see what I called Dr. Bassoe's attention to a moment ago, namely, the prominence of the transverse process of the lumbar vertebra involved in this particular case. See it sticking up there on both sides! The fracture involves the body of the first lumbar vertebra. The back of the body appears to be normal, but it is compressed in front.

When I grasp the transverse process, I can move the whole body of the vertebra, showing how loose that body is and what the danger is of involving the cord by further motion. That vertebra is extremely loose. We will remove the spinous process and laminæ of that vertebra with rongeur forceps, so as to give the cord a chance to retreat backward from the compression by the bodies in front. In that way we will expose the cord freely. There is the cord, you see. There is the deviation. See the depression! See the bone! There is where the cord is in danger. See the level of the vertebra below, and note the difference in the level, which accentuates very nicely the points Dr. Bassoe made, first, that the cord is already compromised, and, second, that there is danger of a further compromise. I am taking out this bony tissue with the very greatest care, because it is here that the cord is most compromised. This is compromising the cord seriously. Now we have it free. There is where the greatest compression was, and there, consequently, the cord was in the greatest danger. Note the difference in the level of the cord here. It is nearly half an inch higher than it is above and below. That's exactly as we figured it when we looked at the second picture. We have removed the bony arch of the first lumbar vertebra entirely, so that the cord is freed from the compression in front. We have done a typical decompression operation of the cord, so that there is no bony arch behind. The cord at the point of fracture is displaced backward at a sharp angle. It is full, round, and resistant to touch above, and flat, flabby, and easily compressible below. I am surprised that he was not paralyzed to a greater degree by this compression.

The muscles will be sutured across the spinal column with catgut. There will be no effort at restoration of the arch or spinous process of the vertebræ which we have removed—that is entirely unnecessary. That does not materially weaken the spine. One would think it would, but it does not. He must be handled with care until the fibrous tissue is fully developed, and in the mean time he will have the necessary support. He will wear a plaster-of-Paris jacket or a leather jacket, which is more comfortable, for a number of months—twelve or fifteen. He should wear a mechanical support to aid in the restoration of bone in the proper position and lessen the callus formation that would be produced without immobilization of the bone.

Now I have sutured this muscle layer with catgut, and outside of that I will suture the lumbar fascia also with catgut, making an accurate apposition. The dura of the cord, you will remember, was not opened. We did not need to expose the cauda or the cord in that position, and we were very careful with the rongeur that took out the transverse processes so as not to contuse the cord.

Let the record show that there was a pronounced luxation forward of the first lumbar vertebra. That that made a sharp angulation, and such a compression of the cord at that point that one could hardly understand why there was not a complete paralysis. It accentuates the great leeway which is normally given the cord in the canal. The spinous processes and laminæ were removed from the first lumbar vertebra, so that the cord was perfectly free behind the zone where it had been compressed, and it can extend backward an unlimited degree now.

That is the treatment for decompression of the spinal cord. It is the same in principle as one treats intracranial tension by decompression. In cases of tumors, and particularly tuberculous granulomata, that occur in the body of the vertebræ and cause injury to the spinal cord, they are often treated best by decompression and not by enucleation or cureting and tamponing with iodoform, as suggested by Sir Victor Horsley. To enucleate tuberculous granulomata from the field in which you operate exposes that field to a mixed infection and the dissemination of the tuberculous process, while if you permit the granulomata to remain encapsulated; if you take out the lamina and the spinous processes, you have made it possible for the cord to spread backward and escape compression. That is an advantage because you know that the tendency of every tuberculous granuloma is to self-limitation.

I recall one case in which we had a very unpleasant experience following the removal of a tuberculous granuloma. We had an infection of the whole field with tuberculosis, finally infection of the cord, and at the end of four months there was a tuberculosis of the dura, a tuberculosis of all the meninges, and a tuberculosis of the cord with permanent paralysis. The patient died. Since that time we have been much more cautious about enucleating a tuberculoma. You have the same conditions in lesions of the spinal cord that you have in the brain. If you permit a tuberculous granuloma of the brain to remain and the intracranial pressure to continue long enough to produce an optic atrophy, that patient will become permanently blind. That blindness can be avoided by a simple decompression—either a subtemporal, parietal, or cerebellar decompression. These operations are entirely free from danger—as free from danger as any so-called minor operation. They are easily done, and through the decompression lessen the intracranial pressure. That stops the destruction of the optic nerve.

We had at one time five children with brain tumor in this clinic. All five responded to the tuberculin test. One was completely blind, one not quite blind, and the others showed various degrees of choked disk. We decompressed all of them. All five lived and recovered from the tumor. They were put under the tuberculin treatment, but we had the professional humiliation of seeing the one that was completely blind remain blind; the one that was almost blind improved a little; the others went on to normal, and fully regained their sight, meaning that they simply had a choked disk and no optic nerve atrophy. This proves that the natural tendency of a tuberculoma is to limit itself, and that the patient will get well under proper treatment. While we did not remove the tuberculomata, every one of the patients lived, and the vision could have been preserved in all if the decompression had been made early in the case of the child that was blind when we first saw it. Fifty-two per cent. of tumors of the brain in children are tuberculomata. There you have a one to two chance of at once doing a decompression and preserving the patient's life and vision. In some of these cases there are no focalizing symptoms, but in all cases of tumors of the brain the symptoms that attract attention early are the vertigo, headache, nausea and vomiting, and the choked disk, meaning an impending optic atrophy. In 100 per cent. of the cases blindness from brain tumor in children, if treated in the early stage of the disease, can be prevented by decompression, while we are waiting for the neurologist to locate the tumor. Never mind the tumor; let that brain expand first. You can do a second operation subsequently. The conditions for the permanent removal of the tumor have not been lessened at all by the decompression, but you have enormously lessened the chances of blindness being produced by the tumor.

It is exactly the same in these cord lesions. Operations done on the cord in a careful way involve practically no risk at all. It is a little more difficult operation than decompression of the brain, which is one of the simplest of all the operations. Decompression of the cord involves a certain amount of definite care not to compromise the cord further.

The skin in this case will be sutured with horsehair, dusted with bismuth subiodid, and sealed with collodion gauze.

[The patient made an uneventful recovery from the operation. He left the hospital within five weeks, wearing a leather jacket. Two months later he returned for examination. He had regained complete control of his sphincters and of all muscle power, except that he could not raise himself on his toes. He returned again after two more months. He could now raise himself on his toes, so that in about five months after the operation he had entirely recovered from his injury. He is still wearing the leather jacket.—ED.]

URETERAL CALCULUS

MULBERRY TYPE AND TUNNELED

HISTORY

PATIENT, a female, single, aged twenty-three, enters hospital on account of recurrent attacks of excruciating pain in the right lumbar and iliac regions. Present trouble began three years ago, following an unusually long walk. That night the patient was awakened with severe pain in the right iliolumbar region. The pain was lancinating, did not radiate, and was accompanied by nausea but not by vomiting. The pain was continuous, with the exception of periods of relief lasting from ten to twenty minutes. The acute pain continued for two or three days, the intervals between the attacks of pain becoming longer, until the pain entirely disappeared in about one week. Patient had no elevation of temperature during the attack, was not jaundiced, and did not notice blood in the urine. These attacks of pain recurred about every three months for the next two years. Each attack seemed to be brought on by some physical exertion, such as a long walk, dancing, etc. The pain invariably came on in the evening. The attacks usually lasted from four to six days, the pain disappearing gradually, and always accompanied by nausea, but no elevation of temperature. For the past year the attacks have come on every three weeks and are shorter in duration, lasting two or three days. The pain is more severe, however, and is accompanied by vomiting as well as by nausea. The attacks are always preceded by a sickening sensation in the abdomen; then the acute pain begins in the iliolumbar region. It does not radiate; the left iliac region is somewhat tender and rigid. At the height of the pain she is unable to extend the right leg; she keeps the thigh flexed on the abdomen and the leg on the thigh. Morphin had to be administered twice to give relief from the pain. The last attack occurred three weeks ago. It began in the evening, with a sickening sensation in the abdomen, followed in a few minutes by pain in the right iliac region, and accompanied by nausea, but no vomiting. She was not jaundiced, and did not notice blood in the urine. She has never had chills or fever.

COMMENTS AND OPERATION

Dr. Murphy (showing the *x*-ray plate): The skiagram shows that the stone is on the right side, about three inches below the kidney, and above the brim of the pelvis. The stone shows fairly well. The metal catheter introduced into the ureter is plainly to be seen, extending from the stone downward into the pelvis. That really makes the diagnosis of stone positive. The stone prevented the further passage of the catheter. What did the urine examination show?

Intern: From five to ten red cells to a one-sixth field. Urine was examined only once. She entered the hospital last night.

Dr. Murphy: How much value have the red cells in just one examination?

Intern: She was menstruating lately.

Dr. Murphy: How lately?

Intern: Last week.

Dr. Murphy: Time enough has elapsed to have the red cells disappear from the urine. There were from 12 to 15 white cells when we examined her at the office two or three weeks ago. She was free from an attack, and was not menstruating at the time. There were only two or three red cells to a one-sixth field. That is enough if you have them examined right. The reason she had these red blood-cells in the urine this morning was that we catheterized the ureter yesterday noon. Why is it that she is free from pain today?

Intern: Probably the catheter which was introduced into the ureter yesterday pushed the stone up.

Dr. Murphy: Why was it that she was free from pain for

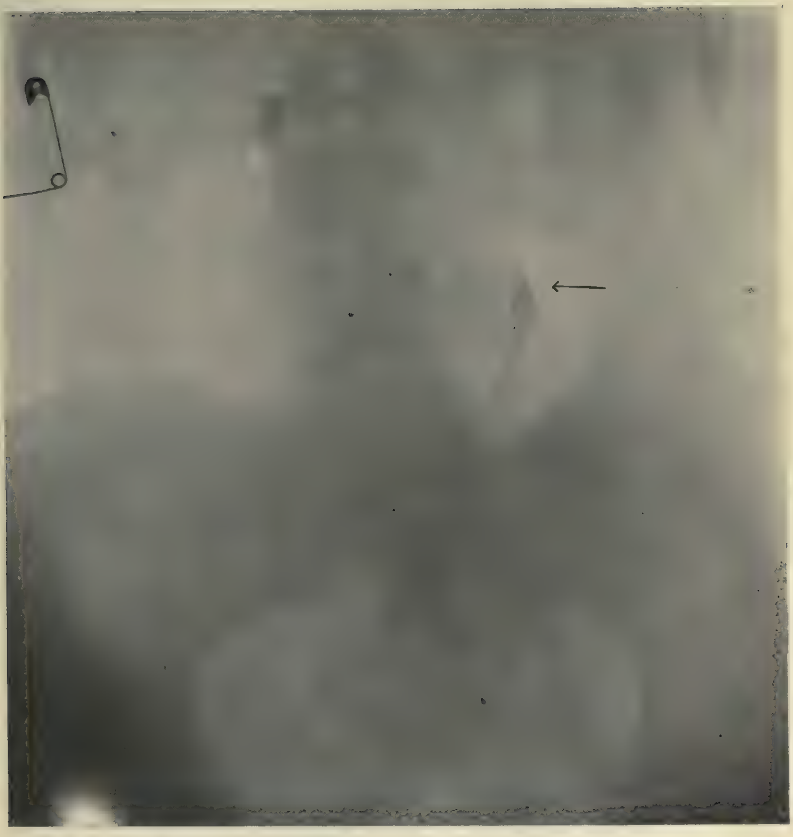


Fig. 54.—The stone is in the right ureter, with the catheter introduced up to the stone—as far as possible. The catheter can be traced downward through the pelvis.

two or three weeks until three or four days ago, when there was no catheter in the ureter?

INTERN: The stone was gradually advancing.

DR. MURPHY: *No, the stone was not advancing,* that is the reason. Pain is the manifestation of the progress of the stone. A stone may rest in the ureter for months, and even years, without causing pain, just the same as a stone can rest in the cystic duct or the common duct without causing pain. When it becomes stationary, it forms a diverticulum in which it is lodged, and the patient is entirely free from pain until it begins to move either up or down. Then these patients have an attack of colic. Colic is a sign that the stone is progressing. If it is arrested suddenly while the tissues are swollen, then they have the colicky pain, the reflex nausea and vomiting, and the reflex pain down into the pelvis, or down into the testicle in the male. One must bear that in mind all the time. The stone has not passed because the pain has ceased: the stone has merely ceased to move forward. The stone may move and the patient have no pain, as was brought out years ago by Dr. Fenger when he said that a stone may move up and down in a dilated tubular structure. When the stone passes out of the dilatation into the narrower lumen the patient will have pain.

This stone is just above the crest of the ilium. I should not have to do anything with the pelvis of the kidney. I may have to bring out the kidney, but it is not my intention to do so. In removing a stone either from the pelvis of the kidney or from the ureter we always cut directly down on the stone. When it is up in the kidney and not very large, we remove it through the pelvis. We rarely ever cut through the cortex of the kidney unless the stone is working its way through there. We have been doing this operation for twenty-one years, and we have never cut through the cortex of the kidney for small stones in the calyces. Dr. Fenger was very reluctant to go through anything but cortex, because he once had a case in which the patient developed a urinary fistula following removal of a stone from the upper end of the ureter, which would not close. The reason it did not close was that the patient had some obstruction below.

Whenever there is an obstruction below, the incision will not close, no matter whether it is through the cortex or through the pelvis.

The patient is placed on the left side, with the right leg drawn up and the left leg extended. A sand-bag is placed under the kidney region or loin. The incision will extend from the outer border of the erector spinæ, one-half inch below the twelfth rib, downward and outward toward the spine of the ilium for a distance of five inches along the anterior margin of the quadratus lumborum.

Now we are in the perirenal cellular tissue and find it perfectly normal. I do not wish to divide the ilio-inguinal nerve. One should not divide it or include it in the closing stitch. There is the lesser peritoneal fatty tissue. There is the kidney. I will go well behind to divide the capsule. The stone ought to be just about two inches below the pelvis of the kidney. See the kidney move up and down in the field with respiration. There is some enlargement of the upper pole of the kidney. We will have to bring out the kidney, so as to give us freer access to the ureter. The pelvis is dilated. There is the ureter. The ureter is dilated just below the kidney pelvis, as far as the stone. There is the stone. I can feel it very well, but I cannot move it backward toward the kidney. It is just about where we estimated it to be—three inches below the pelvis of the kidney and just above the crest of the ilium. Its surface is rough. It is a mulberry stone. That is why I could not move it. I grasp the fibrous layer of the ureter with hemostats, so as to steady the ureter, and then incise its wall directly over the stone, making a longitudinal incision. The stone is removed with a small spoon. It is mulberry shaped, about three-quarters of an inch in length and half an inch wide. It is tunneled. Three stitches will close the opening in the ureter accurately. We use fine catgut. A small rubber tube drain will be placed down to the ureter, and secured to the skin with a silkworm-gut fixation suture. We have a case in the house now with a history very much like this case, but we are unable to find the stone. We can find the colon bacillus, however, which leads us to believe that there is a stone there. You never have a stone form in the normal kidney or in the normal ureter. These suppurations and stone formations are the result of ordinary retentions or obstructions to the normal outlet, often with infections.

Now, then, you know a lumbar hernia is one of the very disagreeable sequences of an operation of this kind. Therefore we will make the closure secure by bringing the muscle wall together with strong catgut. There is the ilio-inguinal nerve. That must not be included in the stitch. I put in the stitch just outside of the nerve, and I do that with each and every stitch until I am sure that the nerve is out of danger. Catching the nerve in the stitch causes a most annoying anesthesia of the skin down to the ilio-inguinal region. The skin incision and muscle aponeurosis will be closed with interrupted figure-of-8 silkworm-gut sutures and a continuous horsehair suture. A large dressing will be applied.

Let the record show that the stone was just about 2½ inches below the kidney and above the crest of the ilium. The kidney was first exposed and elevated, then the ureter was exposed, and the stone was removed. A drain was inserted, and the wound was closed in the usual way.

[The drain was removed after ten days. Primary healing. Horsehair sutures removed. Patient has been perfectly comfortable since the operation.—Ed.]

CEREBELLAR TUMOR

Marked Relief Following Decompression

NEUROLOGIC EXAMINATION BY DR. CHARLES L. MIX

EYE FINDINGS BY DR. RICHARD J. TIVNEN

HISTORY

THE patient, male, aged nineteen, enters hospital on account of throbbing occipital headache, radiating forward to the frontal region, coming on every three hours, and lasting five minutes. Present trouble began six weeks ago, when he first felt a dull pain in the occipital region when he stooped over or lifted a heavy object. These headaches gradually became so severe that the patient stopped working about one month ago. The headaches now came on every three to four hours, and the patient felt so weak that he went to bed. He now also complained of tenderness to touch over the occipital region, and he had to lie on the side of the head to avoid having pain. The throbbing pain in the occipital region now came on every three hours, and lasted five to ten minutes. The pain radiated forward and drew his eyebrows upward. Between these attacks of throbbing pain he now has constant frontal headache. For the past month he has complained of impaired hearing, and on January 17, 1913, he had a throbbing earache in both ears all night. For the past three weeks he has felt dizzy on standing, and has had a marked uncertainty of gait—a drunken stagger. He falls to the left. His eyes are all right as far as sight is concerned. He has lost 20 pounds in weight in the last six weeks. He has had some night-sweats during the past three weeks and also during the day. Previous diseases: Had a cough and "stopping up of the nose" six weeks ago. Had typhoid at nine. Has had no vesical or rectal trouble. His family history is negative. Venereal history is negative. Accidents: Had his nose broken seven years ago. No injury to the head at any time. Has a marked depression in the occipital region.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix, will you please take up the neurologic phase of this case?

Dr. Mix: I saw him four days ago, while he was having one of his five-minute attacks of headache. During the attack the pulse was delayed to 50. The pain in the attack in which I saw him began in the middle of the back of the neck, and spread up over the top of the head. The attack lasted about five minutes. During the attack the patient was quiet, lying on the left side of his head, and rather taciturn. The headache is throbbing. The hearing in the left ear is good. The right ear does not seem as good as the left as to hearing, but he hears the watch at 24 inches with both ears. He had an earache in both ears the night before last (January 18), for the first time since he was seven years old. He has had no earache since.

Physical examination shows the patellar reflexes and knee-jerks normal. There is no Babinski, no jaw sign, no Gordon, and no Oppenheim reflex. The abdominal, epigastric, and cremasteric reflexes are present on both sides and are normal. Sensation is everywhere normal. There are no paresthesias. There is no girdle sensation and no disturbance of the muscle and joint sense, which is perfect in the lower extremities. There is no loss of muscle power. The strength in both arms and legs is perfectly preserved. There are no muscle atrophies.

As to coördination, the Romberg sign (static ataxia) is present. He staggers to the left. Possibly he has a slight left diadokokinesia, and an actual coördination loss in the left leg. He has no ataxia of the left leg as he lies in bed—only on standing. There is possibly a slight weakness in the seventh left cranial nerve, for when he smiles, the left nasolabial fold is poorer than the right. But the occipitofrontalis is equally well moved on both sides of the forehead, and the tongue comes out straight. The ears are reported by Dr. Tivnen as being negative.

There is no cerebellopontile angle tumor, therefore the conclusion is that he has a left cerebellar neoplasm. The rapid onset of the choked disks, mostly on the left side, with a left-sided occipital pain as the storm-center of the attack, with the left-sided cerebellar ataxia, all point to the left cerebellar hemisphere. A pulse of 50 in the seizures denotes increased pressure. A lumbar puncture was not done. The rapid development, with the loss of weight, favors a sarcoma. A possibility of abscess exists, by reason of the rapid course, but the absence of pus in the sinuses and ears and of pulmonary disease removes any possible etiologic factor for cerebellar abscess. Pulmonary disease usually causes abscesses in the cerebrum, rather than in the cerebellum.

Dr. Tivnen: The eye findings in this case are as follows:

Vision: O. D. = $\frac{20}{30}$
O. S. = $\frac{20}{30}$
Position of globe: Normal.
Ocular and lid movements: Normal.
Pupillary—
(*a*) Direct light
(*b*) Convergence, accommodation
(*c*) Consensual
} All normal in both eyes.
Fundi: The fundus of both eyes exhibits a choked disk.
The fields for form and color are concentrically contracted.

The particular ocular manifestation this patient presents, which suggests and confirms the diagnosis of a brain tumor, is the presence of choked disks. The nerve-head of both eyes exhibits this condition, the left optic papilla being more affected than the right. Eliminating the usual causes of optic neuritis in this case, and considering the other subjective and objective findings he presents, the presence of bilateral choked disk is a determining factor in concluding that he suffers from a brain tumor. The presence of this choked disk not only points clearly to the existence of an increased intracranial pressure, due in all probability to some morbid cerebral growth, but it also, in no uncertain manner, calls on the surgeon to preserve the patient's eye-sight by relieving this pressure. Dr. Murphy's decision to perform an early decompression operation on this patient with the object of restoring and conserving his vision would meet with the approbation of every ophthalmic surgeon of experience. Locating and removing a cerebral neoplasm which has produced a choked disk, while important, must, after all, occupy a secondary place in comparison with what should be the prime consideration in such a case, namely—to restore and preserve the patient's visual function.

The usual symptoms of brain tumor are headache, nausea, vomiting, general convulsions, vertigo, and choked disks. Of these symptoms, choked disk is easily the most valuable. It is found in 80 per cent. of brain tumor cases, and cerebral neoplasms are more frequently than any other the etiologic factor in its development. Of all the eye symptoms found in such conditions, it is the one upon which chief reliance must be placed. The value of the symptom as a diagnostic aid in brain tumor is unquestioned. Bramwel tersely expresses it: "The absence of double optic neuritis does not necessarily exclude the presence of a tumor; but the fact that there is no optic neuritis does suggest doubt; and unless the other symptoms of tumor are very clearly defined, or unless the physician feels satisfied that there is no condition present except tumor, which could reasonably be expected to account for the phenomena of the case, he will be wise, in the absence of double optic neuritis, to hesitate before committing himself to a positive diagnosis."

The *modus operandi* of the production of choked disk is by no means a settled question. Von Graefe first described the symptom and its relationship to intracranial disease. His idea was that the tumor caused pressure, either directly or indirectly, upon the cavernous sinus, thus producing congestion of the ophthalmic vein and central vein of the retina, leading to edema of the papilla and hemorrhages. Leber suggested an inflammatory theory under the belief that the intracranial disease leads to the development of phlogogenous material, which passed into the cerebrospinal fluid and so to the nerve-sheath, nerve, and papilla. Inflammation was set up, with edema and infiltration, as secondary manifestations.

The improvement of the choked disk which follows the decompressive operation would appear to emphasize the importance of the mechanical element—that is, the increased intracranial pressure—as etiologic factor of prime consideration in the production of choked disk. At the present time, however, the consensus of opinion is that the question must be held to be "*sub judice.*" In the present state of our knowledge of choked disk the ophthalmologist is only able to assert to the surgeon that the presence of the symptom indicates, after eliminating other causal factors, that the patient is suffering from a brain tumor. Its presence does not aid materially in localizing the neoplasm. While the rapidity of the onset of the symptom, its progress, its marked accentuation on one side, its unilateral or bilateral ocular involvement, may be more or less suggestive in each individual case, yet it is almost always impossible, from these clinical data alone, to arrive at any conclusion of value whether the growth be situated in the cerebrum, in the cerebellum, or in other parts of the brain. Neither does the presence of this symptom nor the stage of its development indicate with any dependable and unvarying certainty the duration, the character, or the size of the neoplasm. Its value in the average case is almost entirely limited to the fact that it points almost invariably, other etiologic factors being excluded, to the presence of a brain tumor. Tumors of the corpora quadrigemina, of the cerebellum, and of the parieto-occipital region are said to show the highest percentage of choked disks; while, according to some authors, pontine tumors and those of subcortical origin comprise the majority of cases in which the symptom is absent. Cerebellar tumors and those in the midbrain and thalamus are reputed to produce a more intense choked disk, with rapid invasion of visual function, than those of the cerebrum, the subcortical, parietal, and frontal regions.

Choked disk is almost always bilateral, though it may differ in intensity on the two sides; a monocular choked disk is rare. No precise dependability, either as a late or as an early symptom of brain tumor, is exhibited by the symptom, as it is exceedingly capricious in its appearance in this respect.

The effect on vision of the choked disk varies considerably. Central vision is frequently normal, and the invasion of this function is usually progressive, and seldom of sudden onset. In this patient the central vision is almost perfectly normal, and yet he exhibits a well-marked choked disk of both eyes. This is not an unusual experience, and the fact that a patient makes no complaint of failing sight should not warrant the conclusion that he does not have a choked disk. The external appearance of the globe gives no clue to the pathologic process within, but appears perfectly normal. The pupillary reactions, in the average case, are likewise in no way suggestive. Examination of the fundus and charting of the fields for form and color are the most dependable procedures in a suspected case.

The prognosis as to sight in a case of choked disk due to brain tumor, in a general sense, depends upon the institution of surgical measures to relieve the intracranial pressure by either a decompression operation or the removal of the offending neoplasm. The earlier these measures are instituted, and the greater the measure of success attending them, the more hopeful is the outlook for restoring and conserving vision. Without such surgical intervention the optic nerve involvement in the majority of cases progresses slowly but surely to optic atrophy and consequent blindness. Dr. Murphy's energetic attitude in supplying this boon to a considerable number of cases coming under my own personal observation has, by conserving their vision, conferred happiness and usefulness where blindness and invalidism were, without the application of his surgical skill, inevitable.

While choked disk is not usually of itself of distinct value in assisting the surgeon to localize the intracranial neoplasm, certain other ocular phenomena may at times be of service to this end. Without entering into a discussion of their relative localizing value, these symptoms include the following:

Hemianopsia or hemiachromatopsia, sensations of light, Wernicke's hemianopic pupillary sign, alexia and optic aphasia, inversion of the color-fields, associated ocular palsies, nystagmus, the symptom-complex of Benedikt, deafness, etc.—of this group of symptoms the alterations in the visual field are perhaps the most commonly present, and more likely to prove of service as an aid in localization of the tumor. In no department of medicine, perhaps, is the coöperation of the ophthalmologist of more value to the patient, to the surgeon, to the internist, and to the neurologist as in this singularly complex and baffling field of brain pathology. From a visual standpoint, the prognosis in our case is encouraging in view of the early surgical measures Dr. Murphy is instituting this morning for the relief of his intracranial pressure.

Dr. Murphy: You also note that this man has a pronounced depression on the back of the head. There is no history and no accident to account for it, so that the condition must be congenital. The question is, can we remove the tumor, and if not, what will we do? If it is malignant, shall we remove it? No, because it will recur. If it is not malignant; if it is a tuberculoma, for instance, that has been coming on for a long time, the thing to do is a decompression. In only about 5 per cent. of the cases of neoplasm of the brain is the tumor ever successfully removed. About 52 per cent. of the brain tumors in children are tuberculomata. The tendency of every tuberculoma is to self-limitation in growth, and all one needs to do is to relieve the intracranial pressure while the tumor is growing, and give tuberculin. This will prevent the optic atrophy and the other symptoms of intracranial plus pressure. But do it early, before the patient has his optic atrophy. Do it when he begins to have the choked disk, the vertigo, the headache, the nausea, and the vomiting. It is too late if you wait until the patient is blind.

This patient's head has been shaved and painted with iodin. I will commence the incision in the occipital region, along the midline, and going around to the left, so that the base of the skin-flap is directly downward. The hemorrhage is checked with hemostats, and later these bleeding points will be ligated. A trephine opening is made in the bone in the center of this area, and the bone is removed with rongeur forceps. As we expose the cerebellum its surface appears to be perfectly normal, but there is no pulsation at all. There is evidently considerable intracerebellar pressure. As I continue to remove this bone the cerebellum is bulged out through the opening, showing the tremendous intracerebellar pressure. As I enlarge the opening this pressure becomes less, and the pulsations of the cerebellum are beginning to return. I have gone upward as far as the lateral sinus. The tentorium can be seen above, and the foramen magnum below. We are out as far as the mastoid cells on the side. Wax stops the bleeding from the bone very effectively. I see no occasion for opening that dura over the cerebellum; there is nothing in it to be removed. The muscular flap will be sutured with catgut, and the skin with horsehair, and a starch bandage or head-cap applied.

We feel that we are going to do this patient a great amount of good by a simple decompression operation. His reaction to tuberculin was negative. That does not mean that he has not a tuberculosis, because encapsulated tuberculomata often do not give a reaction. Tubercular foci that are not encapsulated usually give a positive reaction.

Let the record show that the cortical surface of the cerebellum was exposed from the foramen magnum to the cerebellum tentorium, and from about one-half inch to the right of the midline through the mastoid process; that there was no visible neoplasm and no pulsation; that a palpation was made of the cerebellar pontile angle, and no tumor could be found and no cyst exposed; that the bone was removed and the muscular flap returned; and that the brain was not punctured.

Report of case the morning following the operation:

DR. MURPHY: I went in this morning to see that boy on whom we operated yesterday and asked him how he was. He said: "Fine, this morning," showing how quickly these patients respond when you release the pressure on the brain by a simple decompression operation.

If the lesion is a sarcoma, the patient is going to die. If it is a non-malignant lesion, he is going to live, whether a decompression is done or not. If he lives and you do not do a decompression, he is going to be blind. We had at one time in the hospital five cases of tuberculoma of the brain. We operated on all of them. One patient who had been completely blind remained blind; a patient who had been partially blind had a little improvement; the next patient, who had only a beginning blindness, was greatly improved; the other two made a complete recovery. That shows what a decompression operation can do in beginning lesions of the brain. This man gave evidence that the tumor was in the cerebellar zone. He had the choked disk, great vertigo, and all the other symptoms indicative of a cerebellar tumor.

VISITING DOCTOR: How large does a tumor in the brain have to be before it causes choked disk and optic atrophy?

DR. MURPHY: It depends on the rapidity of growth of the tumor. They grow to a great size often without causing symptoms if they are slow of growth, showing how the brain accommodates itself to the tumor. The rapidity of growth produces the symptoms. It is very much like death due to the loss of blood. The mortality from hemorrhage does not come so much from the quantity of blood lost as from the rapidity with which the blood is reduced in quantity.

[This patient progressed favorably for five weeks. His mental condition had cleared up. He was bright and well. Then he had a severe convulsion and died in six or seven minutes. Necropsy was not permitted.—ED.]

OSTEOMYELITIS OF TIBIA

TRANSPLANTATION OF TEN-INCH SEGMENT OF BONE FROM OPPOSITE TIBIA

HISTORY

Patient, female, aged twenty, comes to hospital on account of an old discharging wound on the anterior surface of the left leg, just above the ankle, and because of inability to bear weight on the leg, due to absence of a considerable portion of the tibia.

In October, 1908, the patient was walking among some cement blocks, when she made a misstep and either contused the inner side of her left leg (region of the internal malleolus), or turned the foot outward forcibly—she does not know just what happened. She was conscious of the injury the moment it happened, but she did not have marked pain. She walked about six or eight blocks before she noticed that her foot was swelling. She went at once to a doctor's office, and by that time the lower part of the leg and the foot were so swollen that the doctor had to cut the shoe off. As soon as the tension was relieved, she began to have pain in the leg and she could not bear her weight on it. She says there was a bruise just over the internal malleolus. The doctor put on a medicated clay poultice and bandaged the leg. She was taken home and put to bed. The pain was quite severe that day and the next day, but the third day she was up in a chair. She does not know whether she had any elevation of temperature. She sat around for two weeks, then got up on crutches and went to school. There was very little pain in the leg during the day, but at night it became much worse and she could not sleep.

She had a plaster cast put on three weeks after the injury (extending from the knee to the toes—toes and heel free). This cast she wore for eight months. The pain was much less severe.

In June, 1909, she took off the cast and walked on the outer side of the foot. She could not put her weight on the inner side of the foot on account of the pain it caused. She walked this way until November, 1909, and then she got a brace that extended to the knee. She got along very well with this brace until May, 1910. One evening in May she had a chill and the following day she was very sick, and had a high temperature. She was in bed two months. During the first month the skin below the malleolus ruptured and the wound discharged pus. Soon afterward a piece of bone appeared in the opening. It was removed. The drainage continued for four or five weeks, and then the wound closed. She began to walk on the outer side of the foot again (July, 1910), and in November she began to have pain in the knee at night. February, 1911, she had pain in the toes. The knee-joint was very much swollen and weak. She had a great deal of pain in the region of the hip-joint.

June 12, 1911, she had another chill and had fever. She was in bed for two weeks, when an operation was performed on the leg. She says when they attempted to chisel a hole through the tibia a little below the knee the shell of bone crumbled, and there was an accumulation of pus found inside. She had three operations up to December, 1911. The lower part of the tibia began to slough off in February, 1912. Ten pieces of bone came away in all. February 24, 1912, the greater portion of the tibia was removed, leaving a stump of bone at the upper end and a small button below of the internal malleolus. She was in the hospital three months. The plaster cast was taken off in August, 1912. She wears a light canvas stocking now.

COMMENTS AND OPERATION

Dr. Murphy: When this girl came to the hospital two weeks ago she had an unhealed patch over the lower end of the tibia. That is healed up completely now, and we are ready to do a transplantation. A very large implant will have to be put in here. The upper articular end of the tibia is intact. The lower articulation is absent except a piece of the astragalus articulation, which is intact. About four inches of the upper end of the tibia

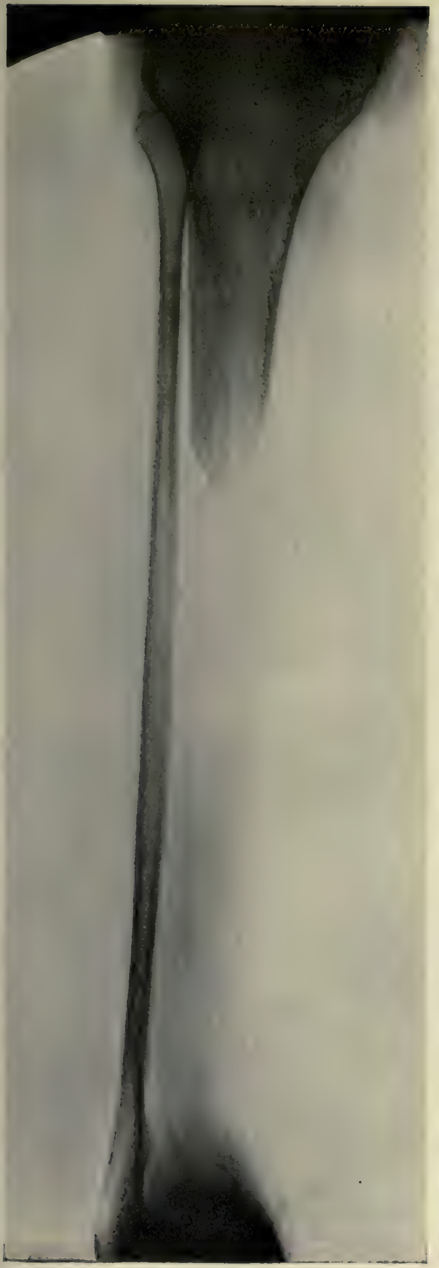


Fig. 55.—Skiagram before operation. Ten-inch defect of tibia, result of osteomyelitis. The lower fragment is very small—just a portion of the internal malleolus. The fibula is normal.

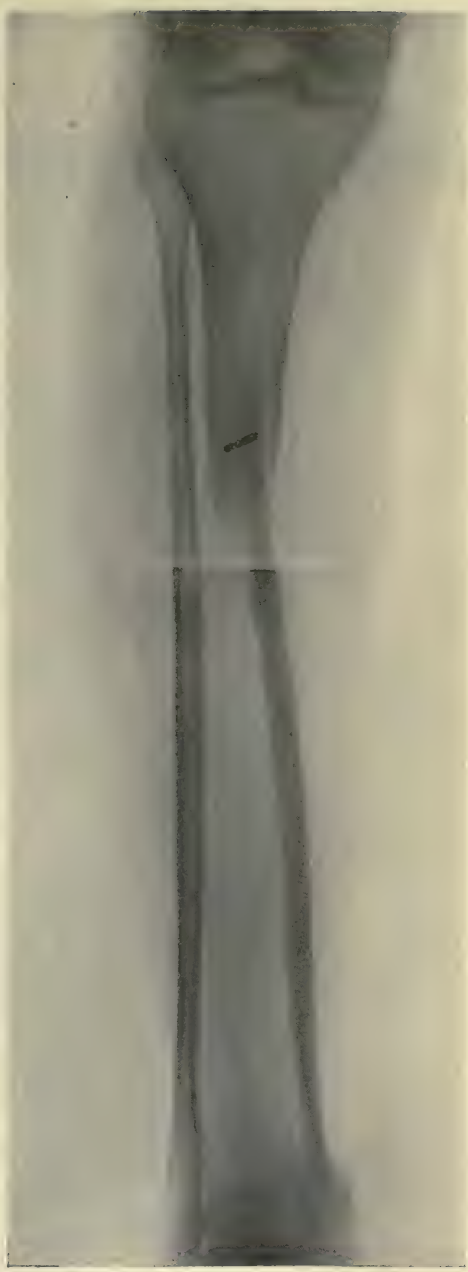


Fig. 56.—Skiagram eight weeks after transplantation. The nail in the upper fragment prevents the transplant from sliding up into the medullary canal. Along the sides of the transplant can be seen new-formed bone. The lower end of the transplant is embedded in the remnant of the malleolus.

remain, but only a part of the internal malleolus remains of the lower end. The fibula appears to be normal. A twelve-inch incision will be made over the tibia, or rather where it should be. The tibialis anticus is intact. The lower end of the upper fragment will be exposed and freshened, and the internal malleolus at the lower end will have its surface freshened, and with the medulla reamer the canal in the upper fragment will be enlarged for an inch and a half. The lower fragment is so small that we will simply make a slight depression in it in which the end of the transplant will rest.

There is not much hemorrhage here because we have put a constrictor on the thigh. Even without the constrictor there is not much hemorrhage in these cases, but we prefer a dry field to work in. The constrictor is always removed before the wound is closed, and all bleeding points are ligated. We also observe the usual precautions against sepsis, avoiding every contact with skin or gloved hands. That is very essential to success in bone work. The edges of the wound are overlapped with towels held in place with towel forceps.

We are now ready for the implant. This leg will be covered with sterile towels while we remove the implant from the crest of the opposite tibia, which is exposed in the same manner as in the case of this leg. We will need a piece of bone 25 cm. long by 1/2 by 3/8 by 5/8 inch. We must be very careful not to break this fragment. It is removed with the chisel. The periosteum will not be removed from this implant.

With a small V-shaped cabinet-maker's chisel we will make a straight groove along both sides of the tibia to the desired depth. Then I deepen the groove with a straight chisel. The crest of the tibia has been cut crosswise with a Hey's saw above and below, marking the ends of the implant. I want to make that bone split in the line of the grooves without fracturing it. We have succeeded in removing this piece in its entirety. It is the longest transplant we have ever made. It will be lifted out of its bed very carefully with two sequestrum forceps, and implanted into the gap in the tissue between the fragments of the tibia of the other leg. The usual dressing of bismuth subiodid powder, collodion gauze, and 5 per cent. phenol gauze is applied. This leg will not be put into a cast.

The implant is now placed in the bed prepared for it. The upper end is driven into the reamed-out medullary cavity in the upper fragment of, and the lower end is carefully placed in the depression in, the internal malleolus. A small nail is driven into a drill-hole made through the upper fragment and the implant so as to prevent it from sliding upward into the medullary canal of the tibia. That completes the operation, except for the closure of the wound.

We will approximate that aponeurosis of the muscle first with catgut. I would like to use phosphor-bronze wire, but its ends cause just enough pricking irritation sometimes to be annoying to the patient. The skin will be closed with horsehair.

In this case most of the osteogenetic force will come from the upper fragment because of the small size of the lower one.

Visiting Doctor: How long approximately will it require for that bone to regenerate?

Dr. Murphy: I would say just about five months. The leg must be kept absolutely quiet. It must be put in a firm cast. The osteogenesis commences above and goes down. If the wound remains sterile,—and we hope it will because we have exercised all the precaution we could,—complete regeneration will take place. I did not touch either an instrument or my glove to any part of that wound or the transplant. Now you will ask what becomes of the transplant. Remember that the transplant is merely a carrier of the Haversian vessels and osteoblasts, and that it will eventually be absorbed, but before it is absorbed, if it remains sterile, it will aid in producing another bone of the same size as the original tibia, so that she will be able to support her weight on it. The bone grows just the same as it does in children during the period of its development and growth. It will finally attain the size required by nature to support the weight of the body.

Visiting Doctor: What value has the periosteum?

Dr. Murphy: I do not know that the periosteum has any value. I do not know that it accelerates the growth of bone.

We know that bone reproduces itself without the periosteum, but I always endeavor to maintain it whenever possible because it may be of advantage. Periosteum is really only a limiting membrane.

I am closing this skin wound with horsehair because it does not cut through. You can leave that in for weeks and months without any disturbance whatever. In that way it has a great advantage over silkworm-gut. You cannot permit silkworm to remain in for any length of time without getting a transverse pressure necrosis. This leg will be placed in a plaster cast extending from the groin to the tips of the toes. That will keep the leg immobilized until union takes place between the upper and lower fragments. It will be cut with the Gigli saw and will not be removed for twelve weeks.

VISITING DOCTOR: How long will it be before she can use the leg?

DR. MURPHY: In a non-united fracture we have them walking in six weeks because you have regeneration along the transplant and you are not depending on the transplant for support. I will not let this patient walk for five or six months, and the cast will be kept on for three months. The leg will not be dressed for four weeks. Then the first dressing will be made and the stitches removed.

We always dress bone and joint cases with the old-fashioned 5 per cent. moist carbolic gauze. We always expect to have an oozing in these cases, and phenol gauze takes care of it and prevents the saprophytes from growing in it. This gauze has been soaked in a 5 per cent. phenol solution at least three days before using. It usually gets well soaked in that time.

Let the record show that the incision was made along the line of the old scar down to the ends of both the upper and lower fragments; that a socket was made in the medullary canal in the upper fragment by means of the reamer and a smaller one was made in the internal malleolus. A piece of bone, 25 cm. by ⅜ by ½ by ⅝, was removed from the crest of the other tibia and inserted into these fragments. A small wire nail above held the transplant in position. The soft tissues were closed with catgut and the skin with horsehair. A plaster cast extending from the hip to the toes was put on. The foot is in an inverted position, and flexed at an angle of 95°. The cast has been cut with a Gigli saw. We never put on a plaster cast without cutting it at once, otherwise we would get a compression of the external popliteal nerve. Afterward we will take off these loose dressings. Then we will put on a smaller dressing and a tight cast and that will immobilize the leg very firmly. That is the principle of all bone work: First, you must transplant bone from the same individual to get uniformly good results; second, the fragment must be contacted with live bone at at least one end; third, you must immobilize it; fourth, the osteoblasts must come from the Haversian vessels of the living bone to which the transplant is attached to form new bone; fifth, it must remain sterile.

[Primary union of the wound occurred. The case progressed beautifully, as is seen in the skiagrams. Bone regeneration is continuing. The leg is firm, and the patient has full control of all muscles. She has not had any pain or elevation of temperature since the operation. The leg is still immobilized in the cast.—Ed.]

FRACTURE OF TIBIA AND FIBULA (LANE PLATE)

Operation by Dr. Philip H. Kreuscher

COMMENTS BY DR. MURPHY

HISTORY

Dr. Kreuscher: Patient, a male, aged thirty, comes to the hospital on account of a fracture of the left tibia and fibula. On the night of December 20th, while crossing the street, patient was struck by a street-car and sustained a fracture of the left leg. He was taken to a drug-store, where a temporary splint was applied. Then he was sent to Mercy Hospital, where the leg was placed in an ordinary fracture-box.

Examination showed the foot in the everted position, with some deformity above the ankle-joint, but no abrasion of the skin. The skiagram showed a spiral fracture of the left tibia, just below its lower third, and an oblique fracture of the fibula, just above the external malleolus. A lateral view showed no anteroposterior displacement of the fractured ends of the tibia.

A reduction of the fracture was attempted, and the leg was placed in a circular cast, extending from the base of the toes to the upper third of the femur. As is customary in Dr. Murphy's clinics, the cast was divided with two Gigli saws, which had been placed on the leg before the cast was applied. Two days later another skiagram was made, which showed that the fracture had not been reduced, and that the lateral displacement of the fragments was greater than when the first skiagram was made. On the advice of Dr. Murphy we concluded to do the open operation, approximate the fragments, and hold them in position by means of a Lane plate.

COMMENTS AND OPERATION

DR. MURPHY: This was a simple fracture put up in a cast. When the cast was taken off, it was worse than before. You can see from the skiagram that there is a little more deformity since the cast was put on than there was before. The fibula is fractured in its lower third. It is ten days now since the accident. Now we are going to put in a Lane plate.

I have had two sets of experiences that contrast very greatly. One was my experience in the Cook County Hospital, where we have the largest fracture service in Chicago. My service here lasted for over twenty years. The other was my service in the Alexian Brothers Hospital, which lasted for ten years, and part of the time overlapped with my county service. There we have the second largest fracture service in Chicago. We had two fracture services under two different conditions. The people who came to Cook County Hospital were always an improvident class of people, underworked and overfed. In the Alexian Brothers Hospital it was just the opposite: we had a class of artisans, overworked and underfed—honest, hard-working people. It took from one and a half to two and a half weeks longer in the Alexian Brothers Hospital to get a union in fractures than it did in the County Hospital, where we had many more failures of union than at the Alexian Brothers Hospital. The limbs were perfectly immobilized in a plaster cast, and the patients put in bed. Those were the cases in which we were most likely to have non-union. Then we came to believe that a certain amount of motion and irritation of the ends of the fragments was necessary in all fractures of the leg. In the last couple of years of my service at the County Hospital I succeeded in reducing the number of cases of non-union by getting the patients up ten days after the cast was put on, and letting them walk around with the cast on. That is the general estimate of the situation, which is very often better than a statistical estimate. Since that time we treat all our simple fractures of the leg by getting the patients up and about right away.

There should be no trouble here in making the reduction.

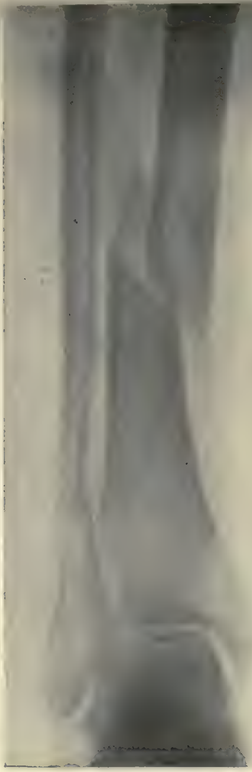


Fig. 57.—Skiagram before operation. Oblique fracture of tibia and fibula. The fibula fracture has united. The upper tibial fragment was displaced backward, the lower slightly overriding. No effort at union.

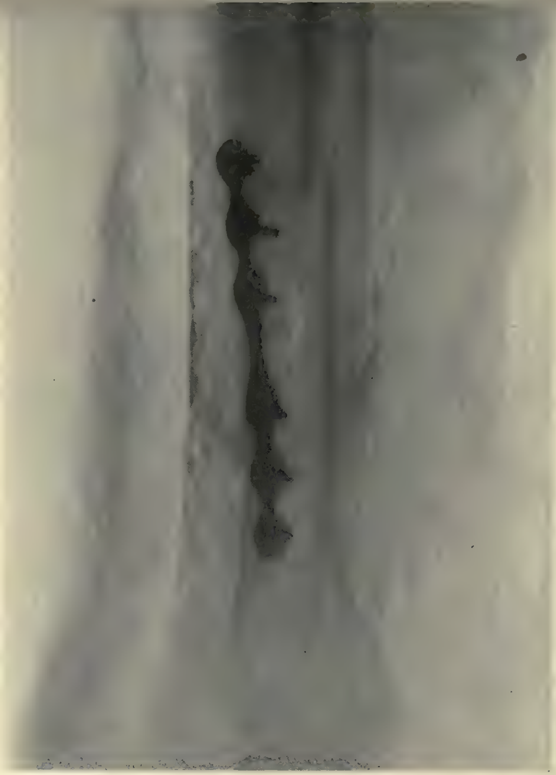


Fig. 58.—Skiagram after operation. Lateral view. Slight displacement of lower fragment to the right (backward), but bony union is taking place.

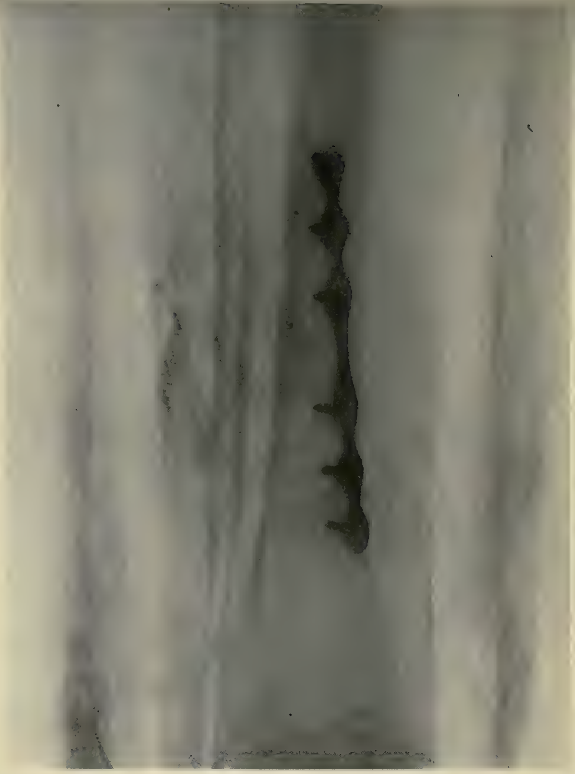


Fig. 59.—Skiagram after operation. Postero-anterior view with six-screw Lane plate maintaining the approximation of the tibial fragments. Perfect alinement of bone in this plane.

The fragments should come right into position. The dangers of infection are enormously enhanced where one has to make the reduction by manual force instead of using instruments. Reduction is often a difficult task. Take a fracture of the femur in a stout man: a great deal of pressure is required to reduce it. That is the greatest source of infection, the manipulation that is required in the effort at reduction.

I am just the referee in this operation. Dr. Kreuscher will do the work, as it is his private case.

DR. KREUSCHER: The day preceding the operation the cast was removed from the leg; all the hair was carefully shaved off, and the entire foot and leg scrubbed with soap and water and washed with bichlorid solution, alcohol, and ether, and a dry sterile gauze dressing applied. Just preceding the operation, after the patient had been anesthetized, the entire foot and leg were painted with tincture of iodin, as is the custom in Dr. Murphy's clinic.

An incision was made through skin and fascia, external to the crest of the tibia. Two heavy towels were then placed, one over each skin edge, and retained by towel forceps. This prevented all contacting with the skin subsequently. The deeper tissues were next displaced inward, thus exposing the fractured ends of the tibia. Inspection showed marked lateral and inward displacement of the upper end of the lower fragment, in which there was a V-shaped groove, into which the pointed lower end of the upper fragment had to be placed. By strong traction on the foot, with inward rotation, and with the aid of two flat instruments which served as skids, the fragments were brought into accurate apposition, where they were held by a large bone-holding forceps of the Lane type. A six-screw Lane plate was next applied, and held in position by six 5/8-inch screws. The skin edges were accurately approximated with a horsehair suture. The incision was dusted with a desiccating powder (bismuth subiodid), and covered with a collodion dressing over which a sterile 5 per cent. phenol gauze and cotton dressing was applied. A plaster-of-Paris cast (as described above) was then applied.

Throughout the whole operation extreme care was exercised not to allow anything but instruments to contact with the wound. No instrument was used the tip of which had had in any way come into contact with the towel on the instrument table, or with the gloved hands of operator, assistants, or surgical nurse.

Dr. Murphy: The apposition is perfect in this case, without ever having touched the fragments with a glove. Only sterile instruments were used. The secret of success in these cases is to keep them aseptic. We had a case here a few days ago in which a plate had been put in. The wound healed up nicely, but when we opened it, there were a few drops of pus around the plate and a non-union. The secret of doing this operation aseptically is never to touch any part of the wound with your gloved hands and to keep the instruments sterile.

I remember a case we had when I was professor of surgery at the College of Physicians and Surgeons. We had the dissecting-room right over the surgical amphitheater. In the case I refer to we did a simple hernia operation. By the time the patient got back to his room he had a temperature of 101° F.; that night he had a temperature of 104° F., and the next morning, 105° F. We removed the stitches, but found nothing but an enormous phlegmon on the side of his thigh. Here was a typical streptococcic infection, and every one blamed every one else for causing it, and finally the blame was laid on the dissecting-room. The case ran on for four weeks. At the end of four weeks I brought the patient into the clinic to illustrate my remarks in a lecture on infections. I described the preantiseptic period, in which I was fortunate enough to have been a student and to have graduated. The antiseptic period began when I was an intern. I described a number of cases of gangrene and of hospital gangrene which was so common in the preantiseptic period. It is said that 79 per cent. of Newell's patients died of hospital gangrene, showing the enormous mortality of that day. I showed my case of infection. After the lecture the patient said to me, "Doctor, did you know that I took care of a patient with gangrene who died three days before I came into the hospital?" That told the story of his infection. He had been nursing a man with gangrene, and he was saturated with these microbes. He came into the hospital three days after the death of the man he was taking care of, was operated on, and immediately got the infection. That proved that the infection was not due to the proximity of the dissecting-room, although the dissecting-room was very much like the other dissecting-rooms of that period. The cadavers were not kept sterile, and the room was not kept clean; still we did not have any infection.

Let the record show that there was an overlapping of the end of the upper fragment over the lower. It was "V" shaped. By careful manipulation with the retractor and periosteotome the fragments were brought into perfect apposition, and maintained with a six-screw plate. There was not a deviation of one-thirty-second of an inch when the operation was completed. There was no contacting of the instruments or the gloves with anything. The operation was thoroughly aseptic.

PERIOSTEAL SARCOMA: AMPUTATION OF THE LEG

HISTORY

PATIENT, a female, aged forty-six, enters hospital on account of a swelling just above the left knee. Six years ago patient first noticed a slight swelling above the knee. She had sustained no injury previous to this time, and had no chills or fever, but complained of severe pains radiating from left hip to knee. These pains had preceded the swelling by several months. The pains were shooting and cutting in character, radiated from the left hip to the knee, and the case was diagnosed as one of "sciatic rheumatism." The swelling was tender to the touch, but not red. It was soft, not movable, apparently attached to the bone. The overlying skin was not fixed. It attained the size of a hickory-nut in about four weeks, and then gradually disappeared. It reappeared at intervals of from five to six months, and usually disappeared in about three weeks. This condition continued for four years, when the "sciatica" ceased abruptly one night and the swelling did not reappear for a year. Patient was perfectly well from this time on until one year ago.

About that time the patient stumbled and fell on her left knee. Immediately after the knee felt very painful, but no swelling appeared. The severe pain continued several days, and the knee became weak. The acute pain disappeared, but the soreness remained. The knee became weakened, and the patient stumbled and fell several times, always striking her left knee. After each fall the acute pain returned and continued for several days. The knee became progressively weaker, and the patient stumbled and fell more frequently. Last July (1912) the patient noticed a gradual, symmetric enlargement of the knee, with the area of greatest enlargement just above the patella. The swelling came on gradually, and the knee was very tender. A dull, aching pain was constantly present. The swelling would occasionally diminish in size, but never disappear entirely. Then it would increase in size, and be larger than before. This condition continued until twelve weeks ago, when the swelling increased to its present size, and the knee became red, painful, and very tender. An extension was put on (3 pounds), which relieved the pain completely. The extension was kept on about ten days, and when it was removed, the knee was stiff and motion was limited. At present the swelling is spindle-shaped, measuring about 8 inches in length by 5 in width. It is not painful, but is tender to pressure above the patella.

COMMENTS AND OPERATION

DR. MURPHY: When did the swelling first appear?

INTERN: Six years ago.

DR. MURPHY: When did the swelling appear the second time?

INTERN: In July, 1912.

DR. MURPHY: That history extends over a longer period than is usual in cases of this kind. She had the initial onset six years ago; then, after a while, she went along very well until she had an accident. Following the accident she began to have severe pain in the leg and knee after exertion. The skiagram shows an excavation of the anterior surface of the femur, just above the condyle. A tumor or mass occupies a considerable area just anterior to this excavation or depression. The shadow is rather faint, but is none the less definitely outlined. It looks as though the tumor had originated in the periosteum and not in the bone. The extension of the mass out into the soft parts is the point that aids us in making a diagnosis. The joint is not involved. The tumor has not passed through the head of the femur into the joint.

This may be one of two things—either a sarcoma or an osteitis fibrosa cystica. An osteitis fibrosa cystica rarely occurs at this age—this woman is forty-six. An osteitis fibrosa cystica occurs in children at the age when sarcoma is common. In the majority of cases of sarcoma of bone the limb must be amputated, while

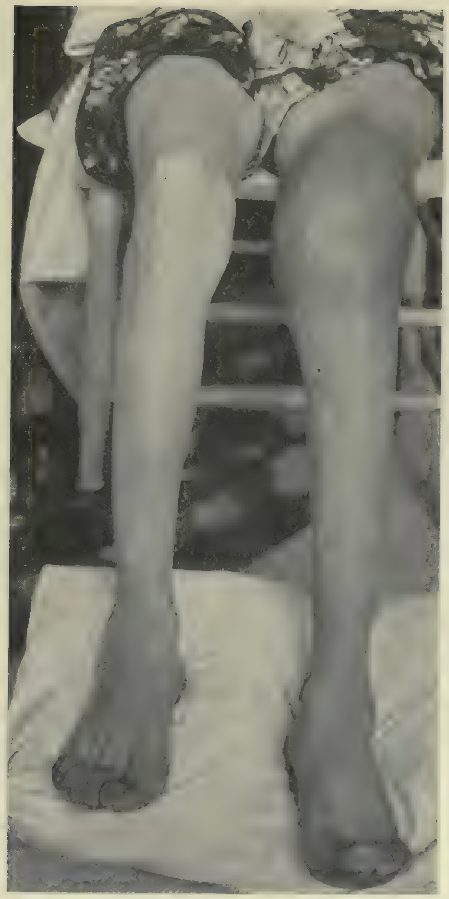


Fig. 60.—Periosteal sarcoma. Anterior view of the leg, showing the symmetric enlargement of the knee.

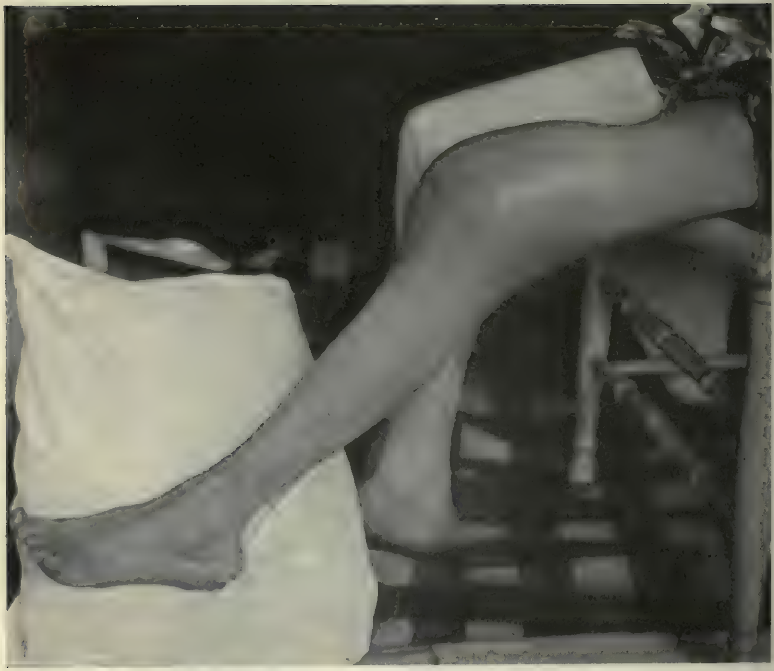


Fig. 61.—Periosteal sarcoma. Lateral view of the tumor.

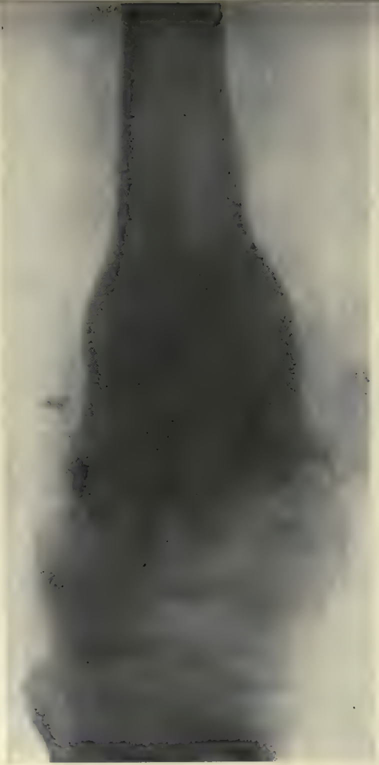


Fig. 62.—Periosteal sarcoma. Skiagram. Anteroposterior view of left leg. The tumor is seen just above the knee-joint, involving the condyles, but not the joint itself. The mottled appearance of the mass suggests that it is breaking down.*

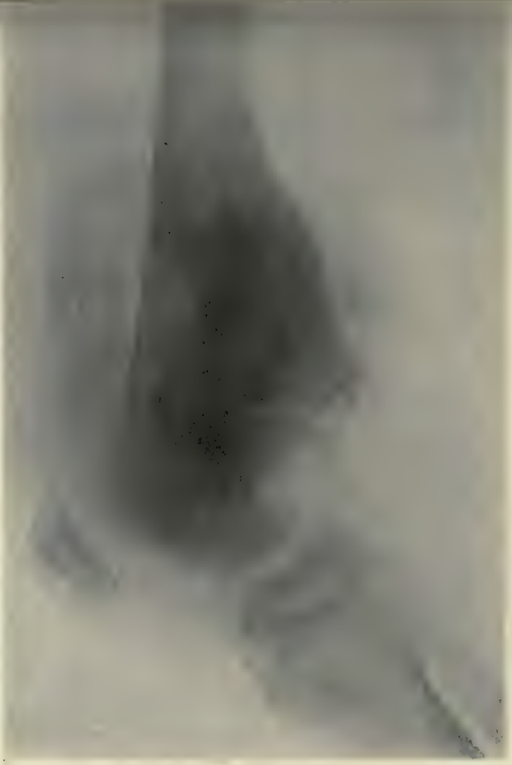


Fig. 63.—Periosteal sarcoma. Skiagram, lateral view, showing the excavation of the femur just above the condyles, with extension backward of the tumor into the popliteal space. The indistinct hazy shadow in front of the bone is cast by the tumor.

in the case of osteitis fibrosa cystica all that needs to be done is to remove the diseased bone and implant a piece of bone from the other tibia. In the latter there is no involvement of the adjacent soft tissues in this disease. There is no periosteal inflammation, which is so pronounced in this case today. In this case the disease has extended into the soft parts and has caused an infiltration of the soft parts, which never occurs in an osteitis fibrosa cystica.

In reviewing this case I do not take into consideration the possibility of removing the tumor and transplanting a piece of bone from her other tibia, as I would if the disease were of the other variety—notwithstanding her age and the length of the bone that would have to be removed. I would not be justified in transplanting a piece of bone in this case, as we have done so often in other cases not malignant, even to the extent of spanning a gap of 10 inches, showing that there is no length which the regenerating bone does not span and reunite if it is contacting with living bone at one end.

We are going to do an amputation in this case in the upper third of the thigh. We are not going to put on a constrictor. This woman has a phlebitis extending above Poupart's ligament, and well up into the iliac vein. If we put on a constrictor we might force an embolus well up into the veins, and from there into the general circulation, which would be fatal to her. The operation will be accompanied by more loss of blood than is usual, but that will not materially injure her. This is an unfavorable case, first, because there may be metastases in other portions of the body; second, because of the phlebitis. Fortunately, she had a cold when she came into the house and we could not operate. Then she got the phlebitis. You see if she did not have the cold, the operation might have been fatal because we would have operated on her immediately, and the phlebitis which was present, but which had not yet manifested itself clinically, might have caused her death. Of course, if the operation terminates fatally now we will know the cause.

The diagnosis in this case is periosteal sarcoma primarily, involving the bone secondarily. We are going to make the posterior and anterior flap amputation from without inward, ligating the vessels as we come to them. There is less danger in this case from hemorrhage than from the passage of the embolus into the general circulation. We will cut through the skin first, then through the muscles a little higher up, and then saw off the bone on a still higher level. This tissue is very edematous. As we cut the veins we see the clots in them. That is why we are anxious. On section, the bleeding from the distal side stops very quickly, because of the diminished lumen of these veins. It is just about as we estimated it, but we chose the lesser of the two evils when we decided on the amputation. We will start the intravenous injection of normal saline solution at once. We are down on the femoral artery and vein now, and the sciatic nerve. We will put two ligatures on the artery and traumatize it on the proximal side of the upper ligature by compression with a hemostat. The purpose of traumatizing the artery on the proximal side of the ligature was emphasized by me in my original article on arterial ligation and suture, published in 1896, when I made the first successful end-to-end suture of an artery on a human being. The double ligature was first suggested by Senn as a means of favoring clot formation. That is not our purpose. We found in our experiments that if one traumatize the artery on the proximal side of the ligature, the intima is fractured and immediately a clot forms in the vessel at that point, blocking the artery and preventing secondary hemorrhage.

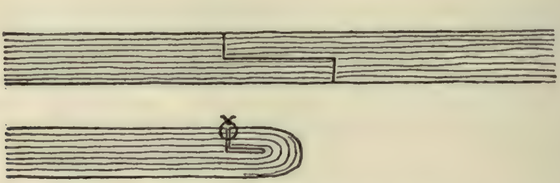


Fig. 64.—The upper illustration shows how the nerve is divided. The hair lines represent the axons. The lower illustration shows how the end of the stump is turned up to contact with the upper cut end and effect axonal contacting.

Next is the section of the sciatic nerve. We do not want to compress that nerve, and we must prevent the formation of an

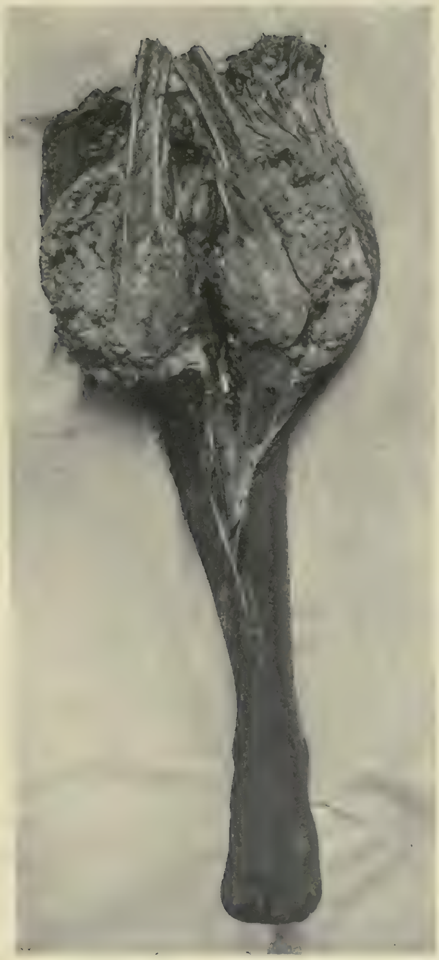


Fig. 65.—Periosteal sarcoma. The leg bisected after amputation. The encapsulated tumor mass on the anterior surface is the primary tumor starting in the periosteum. The bone involvement is secondary. The joint surface of the femur is intact.

amputation neuroma. I will cut this in an L-shape. See how quickly she responds to irritation of the nerve! I will turn up the cut end of the nerve and suture it into the upper angle, so that we will get an axonal contacting of the cut ends. By doing that we avoid the formation of a neuroma and the annoying neuralgias that follow amputation when this precaution is not taken. See how these axons come together end to end! A few fine catgut stitches give us the apposition we desire, and that means so much to the patient.

We are now ready to close the wound. The muscles and fascia are united with plain catgut, making the so-called "Stiegennaht," or "terrace" suture, of Neuber, of Kiel, which makes an accurate apposition of all structures and obliterates dead spaces effectively. The superficial layers of the wound are approximated with silkworm-gut. These sutures are really tension sutures. The skin edges are united with horsehair. A guttapercha drain is passed in at one angle of the wound and out at the other. It is secured to the skin with a fixation suture of silkworm-gut. A 5 per cent. moist phenol gauze dressing is applied, and over this a large gauze and cotton pad held in place with roller bandages and adhesive straps.

One never likes to do an amputation. An amputation is always an admission of defeat. We have no tension at all on the skin-flap. There will be more oozing in this case than occurs in the average case. We put on more ligatures than we usually do for the same reason.

[Examines the specimen.]

This was a sarcoma. A sarcoma that occurs in bone, and of the small round-cell variety, is the most common next to the giant-cell variety and is the most malignant. It more frequently occurs as the sequence of trauma than from any other condition. The trauma is always the initial cause, and is always of mild severity. This sarcoma evidently began in the periosteum and then spread to the bone. The tumor is distinctly encapsulated. It is breaking down in its center. One recognizes that at once as sarcomatous tissue. You can see how disadvantageous it would have been to do a bone transplantation in this case.

There is an enormous deposit of sarcomatous tissue on the anterior surface of the femur, and still a portion of the bone is intact and the joint is not involved at all, showing how slow the sarcoma was to attack the cartilage. It went through the epiphyseal line, but did not penetrate the joint cartilage. It is not a chondrosarcoma, which is not so malignant as the small round-cell sarcoma. Notwithstanding the fact that it is encapsulated, one is always fearful that it has metastasized in some other portion of the body. A carcinoma practically never metastasizes through the blood-vessels: it usually spreads through the lymph-vessels. A sarcoma practically, as a rule, metastasizes through the blood-vessels. That is the difference between the two—one usually travels through the lymphatics and the other through the blood-vessels. If you had an encapsulated carcinoma like that, you would feel that you could take it out and not have a recurrence. The tumor-cells in this type of sarcoma are carried to other parts of the body by the blood, and secondary growths appear, especially in organs containing terminal or end-arteries, such as the lung, brain, spleen, and kidneys.

We will not be able to tell for two or two and a half years that metastases will not form in this case. This bone could be used as a transplant, but we have no patient in the house needing a transplant from whom we cannot get a sufficient quantity of bone from himself. We never take it from another individual if we can take it from the same individual, because auto-transplants give the best results.

What this lesion was primarily I do not know. What her first trouble was I do not know. I think it is a sarcoma that grew very slowly. These tumors contain giant-cells. They are often called giant-cell sarcomata. They are the least malignant of the sarcomata. The least malignant of these is the tumor that occurs first on the gum, the epulis; next in the turbinate, the frontal sinus, and the sphenoid. These are the four positions in which you have the giant-cell sarcoma of least malignancy. They recur and recur. I remember one patient I saw in Berlin who had been operated on 118 times for one of these sarcomata. Each time the doctor had taken off the tumor but left the periosteum from which it originated. It recurred and recurred until v. Bergmann divided the nose, split it up into the frontal bone, and took out all the periosteum. Then he said it would never recur.

When the tumor originates in the alveolar process, it is not sufficient to remove the tooth—the membranous lining of the alveoli must be removed. I remember when I was a student it was customary to extract the tooth and take out all the alveolar process, so as to obviate a recurrence. If you merely take out the tooth, it is a question whether the patient will live long enough for you to take out the epulis 118 times.

The type of cell has much to do with the malignancy, also the alveolar arrangement of the cells. When I was a student they laid great stress on this alveolar arrangement. Now it is the type of cell only that is considered. The small round-cell sarcoma is the most malignant. That type of tumor occurs in people under twenty-five. It usually follows a trauma, and is associated with an elevation of temperature, and many times with general systemic intoxication, very closely resembling the clinical picture of an infection. Next in malignancy is the large round-cell sarcoma; then, third, small spindle-cell tumor; fourth, a combination of these two, and fifth, a combination of all of these.

In the early stage of the small round-cell sarcoma it is difficult to tell from the clinical manifestations whether it is a malignant growth or an infection, on account of the severity of the constitutional and local symptoms. One day, along in 1880, when I was just about to leave the Cook County Hospital, I saw my first severe case of this kind. The patient was a stevedore. He was carrying some planks out onto the dock from a vessel, when he slipped and landed squarely on his knee. He worked the remainder of that day and the next day. That evening he had a high temperature. He came into the hospital on the morning of the third day with a temperature of 104° F., and with the knee swollen and tender. He became delirious, and had all the symptoms of an acute bone infection. Dr. Fenger, who was my attending surgeon at the time, examined him. He did not know whether it was a sarcoma or an infection. He advised waiting a few days. On the fifth or sixth day he began to get the pulsation—the typical pulsation of an aneurysm associated with the round-cell type of sarcoma. Then it was comparatively easy to make the diagnosis, because the swelling was extensive, involving the whole lower end of the femur. Dr. Fenger amputated the leg just below the trochanter. He removed all the bone that was involved, but retained a small upper fragment, because it would aid in wearing an artificial limb. That patient left for Germany immediately after he recovered from the operation, and we never learned what was the final outcome of his case, but Dr. Fenger believed that he would have a return of the disease, and we believe he was right. The dissection of the specimen showed that it was a large aneurysmal sarcoma of the small round-cell type. Great bloody caverns had formed in it in this short period of time. The high temperature in this case tended at that time to make one believe that the sarcoma was of infectious origin.

Another type of sarcoma is the slowly advancing chondrosarcoma, often containing giant-cells. That tumor is less malignant than the other sarcomata, and rarely metastasizes through the circulation. That is the type in which you can remove the bone involved and implant in its place a piece of the patient's tibia from the other limb. We have had two such cases, and the results have been splendid. One was a case of sarcoma that developed in the trochanter as the result of a fall. We decided to make an excision of the tumor and transplant a portion of his tibia. We did that, and sutured the muscles around the transplant, but in putting in the fragment we split it accidentally. There was no other place from which to take a new fragment, so we wired the two pieces together. Fortunately, this gave us a fairly good trochanter, and the results were splendid. Notwithstanding that one of these fragments only contacted with the split portion of the other fragment and not with living bone, the upper end of the femur is beautifully regenerating. It is now seventeen months since the operation. The man has good motion in his hip and is allowed to put most of his weight on his leg.

The second case was one of chondrosarcoma of the upper end of the tibia, not involving the knee-joint. We removed 5 inches of the tibia, leaving the articular surface, and transplanted a piece of bone taken from the crest of the other tibia. That was done fifteen months ago. The result is splendid. The bone is regenerating rapidly, as can be seen in the skiagrams.

[The drain was removed on the fifth day. Primary union; little discharge; patient very comfortable.—Ed.]

CHRONIC TROCHANTERIC BURSITIS

HISTORY

Patient, a male, aged twenty-six, enters hospital on account of lameness and pain in and discharge from the right hip. Present trouble dates back fourteen years (1899), when a rock, weighing 30 pounds, fell on his right hip. This injury caused severe pain in the right leg, but he was not incapacitated. The following day he went to school and suffered no inconvenience subsequently, excepting that during changes of weather he had slight pain in the region of the right hip. Eight years following the accident (1907) patient noticed a small swelling over the right hip (trochanteric region). This mass was freely movable, was soft, and was not painful or tender to touch. It gradually increased in size, and after five years (1912) it was as large as a base-ball. During these five years the patient lost 30 pounds in weight. He was then operated on. The tumor was excised. It was soft and had a fatty appearance. A gauze drain was inserted and removed in four days. Patient now began to regain his lost weight, but three months after the mass was excised a second similar mass appeared in the same location and patient immediately began to lose weight again. The mass steadily increased in size, and four months later was about the size of a chicken's egg. Last August (1912) plasters of zinc chlorid were applied to the mass, and in two weeks it disappeared. The skin ruptured, and a yellowish discharge followed. The discharge continued up to October 4th, when the femur was exposed and cureted. A rubber drain was inserted, passing through the thigh muscles. Yellowish discharge in small quantities appeared, and has continued up to the present time. On December 19, 1912, patient, following exposure to cold, had a chill, followed by fever. The following day a pocket of pus was opened in the thigh, and the fever subsided. At present there is a rubber drain in the wound with very little greenish-yellow discharge coming from it. Patient does not complain of pain or tenderness in this region, and has full motion in hip-joint. Has never had cough or night-sweats. Previous history negative.

COMMENTS AND OPERATION

DR. MURPHY: What is the matter with him, doctor?

INTERN: He has a bursitis.

DR. MURPHY: What leads you to think he has a bursitis? May he not have a tuberculosis of the hip?

INTERN: The trouble appeared following the initial trauma. He was not incapacitated for eight years following the trauma. He then noticed a small mass on the right hip, which was fairly movable and not tender.

DR. MURPHY: Might that not have been a cold abscess?

INTERN: He had no elevation of temperature.

DR. MURPHY: It is not necessary that he should have elevation of temperature with a cold abscess; in fact, it is the rule not to have a temperature elevation.

INTERN: The mass was not tender.

DR. MURPHY: A cold abscess is not tender. That cold abscess came from a blow fourteen years ago. He had no limitation of motion; he could put his limb into any position without pain or discomfort, meaning that he never had a tuberculosis of the bone near the neck or close to the joint. He might have had a trochanteric tuberculosis with a cold abscess resulting from it, which would not involve the joint. This mass first increased in size eight years ago—more than five years after the trauma. Where did he have the pain?

INTERN: On the outside of the right hip.

DR. MURPHY: The mass was removed about a year ago. What sort of a mass was it?

INTERN: It was soft and had a fatty appearance.

DR. MURPHY: Where?

INTERN: On the inside.

DR. MURPHY: What kind of a fluid was discharged?

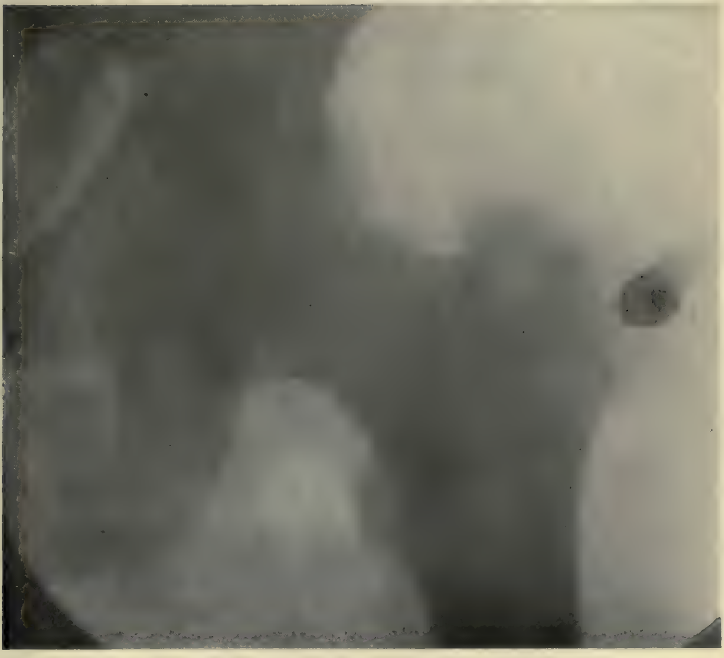


Fig. 66.—Chronic trochanteric bursitis. The mottled shadows in the area of the great trochanter and the irregular shadowy contour of the bone margin from the tip of the trochanter downward and to the right are evidences of disease. The splotch to the right is the drainage-tube in the sinus.

Intern: A yellowish fluid.

Dr. Murphy: It contained a yellow fluid and had fat on the outside. This history is almost a perfect word-picture. It is classic in every detail, and it should be recognized by every one who reads it, and a diagnosis made of the primary lesion that was present when the patient was operated on. Three months after the mass was removed a second mass appeared in the same location. The mass continued to increase in size, and four months later it was the size of an egg. How was he treated, doctor?

Intern: Zinc chlorid plasters were put on, and in two weeks the mass disappeared, but a yellowish discharge appeared and continued until the mass was cureted. A rubber drain was inserted, and the wound has continued to drain freely up to the present time.

Dr. Murphy: Now, then, what does that mean? When this mass was opened, there was no bony lesion of the femur at all, because there never is. The reason they cureted the femur was on the supposition that the lesion was a tuberculosis of the femur, which it was not. It is the classic history of an infection of the trochanteric bursa—always slow in advancing, very rarely appears rapidly, and very rarely appears as an acute metastatic infection following a trauma. The swelling increases in size steadily until it extends half-way down the thigh.

Aspiration and pressing out the contents of the abscess do not do in these cases. It does not suffice to put in a drain. Cureting the bone does not effect a cure. The only treatment is excision of the bursa. Nothing else is worth while. Applications of nitric acid or zinc salve do not cure it. Occasionally, a patellar bursa is similarly infected. It becomes an edematous and thickened mass, and looks like a tuberculous lesion. It recurs and recurs, unless you make a clean-cut dissection of the bursa.

The fluid that is discharged is collagenous in nature. The slowly developing collagen forms a membrane resembling the synovial membrane, covered with flat cells of the cicatricial type, resembling endothelial cells, but they are not endothelial cells. The fluid resembles the synovial fluid, but contains collagen.

I will make a complete dissection of that bursa, and clean the whole thing out. The skiagram shows the infection of the trochanter. It shows where the trochanter was cureted. There is no evidence of a primary disease in the trochanter. Whether there is a secondary disease in the trochanter now we do not know. The management of these cases is the same as that of a postsacral sinus. In a case of postsacral abscess, first dissect out all the sinus and go on down until you come to the abscess. The sinus will go down almost straight, and then branch off, and unless you excise all the branches, in about six or eight months it will discharge again. The epithelial-lined tracts in there must all be dissected out—nothing else will effect a cure. It must be an extremely radical dissection, and all the sinuses must be taken out.

This bursa formation is due to the absorption of fat from the fatty tissue and the development of collagen. In the development of new joints it is the application of this principle that gives us the movable joints. The result of this degenerative or absorptive process in the fatty tissue, with the hyperplasia of the connective-tissue element, is the hygroma. These hygromata are lined with flattened, endothelial like cells, but do not contain fluid under normal conditions—only under pathologic conditions.

Over twenty years ago unsuccessful experiments were made on the external surface of bones and joints in order to get movable joints. Then came the knowledge of the development of the hygroma from the fat, and the fluid which formed in the false joint following the failure of a bony union. We know that in certain cases of failure of bony union following fracture a fairly well-formed false joint is produced, with cartilage on the ends of the bones and a tissue surrounding them which resembles the fibrous capsule of a normal joint. Occasionally, this false joint contains a fluid resembling the synovial fluid. Sometimes there is a blood-clot between the two ends of the fractured bone, with a fairly well-developed capsule surrounding it. It is that knowledge which encouraged us to perform arthroplasties to produce movable joints in ankylosis. It is merely the application clinically of what nature does automatically. If you cannot do that, you are not a very good technician.

We will make a straight incision over the trochanter, on the iliotrochanteric line, and we will excise the old scar. There is the pocket of the bursa. There is the tissue resembling the granulation tissue which is lining the bursa. There is a second pocket lower down, showing how these cases recur. There is a third pocket here at the tip of the trochanter. We will dissect out all these pockets. The spot on the surface of the bone where the last curetment was made is plainly visible, and it is also lined with granulation tissue. We must take all that tissue out, otherwise there will be a recurrence of the trouble. It is very difficult to get all this tissue out. There is so much of it and these pockets branch off in every direction.

It is only the fire of experience that really makes one do a clean-cut dissection in these cases. Now we are down to the place where the trochanter is exposed. There is a pocket under the trochanter. That is where the curetment was done. This operation is more difficult than an arthroplasty. It certainly is more tedious. We will curet the bone and remove all the granulation tissue. The wall of this sinus is one-third to one-half inch thick in some places. It consists of fat and new formed fibrous tissue.

Let the record show that it was a typical case of bursitis which had burrowed in all directions; that we carefully followed each separate sinus to its termination; that we took off the surface of the trochanter down to the normal bony tissue; that the very extensive burrowing of the infection which we found justifies the stress we laid on thorough dissection.

A curetment does no good in these cases. They require a careful, clean-cut dissection. We will put in a small tube drain here and close the wound with deep catgut sutures to obliterate dead spaces, superficial silkworm-gut tension sutures, and horsehair skin sutures. A moist 5 per cent. phenol dressing will be applied.

[Cultures made from the pus before operation showed B.

pyocyaneus, which accounted for the green color. The wound discharged a green-colored pus freely for two weeks, sometimes as much as a pint daily. The drain was then removed, and three weeks later the wound had closed completely and the patient left the hospital. He was given four or five injections of autogenous pyocyaneous vaccine, and it acted like turning off a faucet. The discharge ceased and the man went on to a complete recovery.—Ed.]

LATER NOTE ON CASE OF CEREBRAL DECOMPRESSION DESCRIBED IN FEBRUARY, 1913, NUMBER (p. 131)

Dr. Mix: Mr. Parker has remained in the hospital for some weeks. Following the operation there was for a few days some increased clumsiness in the right arm, with inability to make generalized coördinate movements. There was also for several days, if anything, an increase in his dysarthria. After a few days this dysarthria and spastic paresis in his right arm disappeared. At the present time (January 21) he has recovered and the wound is entirely healed; the size of the opening which was left from his first injury is considerably smaller. He can talk with perfect ease and use his right arm better than his left.

[The patient returned a week later, just before leaving for his home in Arizona. He was entirely well, except for a slight hesitancy or slowness of speech when crowded. There were no motor symptoms and no headache. He said he felt well and strong.—Ed.]

RECURRENT APPENDICITIS—RETROCECAL APPENDIX, WITH DESCRIPTION OF DR. MURPHY'S PROCTOCLYSIS

HISTORY

Patient, a male, aged twenty-four, enters hospital on account of severe cutting pain in the epigastric region. At 7 o'clock yesterday morning patient was suddenly seized with acute pain in the epigastrium, radiating across abdomen and encircling the upper lumbar region. He did not feel nauseated and did not vomit. Had no elevation of temperature, but in the afternoon his temperature was 99.2° F. Pain seems to be aggravated by breathing. Patient is habitually constipated, and has taken an enema every morning for the past four weeks. Considerable flatus is always expelled with the feces. He also complains of slight abdominal distention, with belching of gas about one-half hour after eating. Epigastric region is tender. Has had four similar attacks, the first one three years ago, and the last one six weeks ago, when he was confined to bed one week. Has been constipated for past four years. Has never been jaundiced, and has never noticed white or light-colored feces. Last night he vomited, independent of food or medication. In the preceding attack he vomited a bile-stained fluid following the onset of his pain. Leukocyte count at 8 o'clock last night was 22,000.

COMMENTS AND OPERATION

Dr. Murphy: When did the attack begin?

Intern: Yesterday morning.

Dr. Murphy: What do you mean by "having had similar attacks"?

Intern: He had attacks of pain at different times, but had no nausea or vomiting until last night.

Dr. Murphy: This time he did have nausea and vomiting. He vomited twice last night. He vomited without food or medication. You know in these cases the patient frequently vomits after taking castor oil or peppermint, or whatever is given to relieve the pain, but this man did not have any medication. Did he vomit in the other attacks?

Intern: The last attack was six weeks ago and he says that he also vomited at that time.

Dr. Murphy: What time in the attack did he vomit?

Intern: He had the nausea and vomiting immediately after the onset of the pain.

Dr. Murphy: That is an important point. It is now twenty-six hours since the onset of the pain. Last night he would not let me palpate his abdomen, and he insisted that he had no pain. His father died of appendicitis two years ago, but the case was let go for ten days before any effort was made to remove the appendix. Death resulted from a subphrenic abscess on the right side. His leukocyte count was 22,000 last night; it is 24,000 this morning. His abdomen is very tense, but I could put my finger on a point of distinct tenderness this morning. Last night he had a temperature of 101° F., but that was after we saw him. His leukocyte count is high, in spite of the fact that he has only a slight elevation of temperature.

We have had in all our experience only three cases of acute appendicitis in which there never was any elevation of temperature. Two of these cases occurred within the last two months. One patient had a high leukocyte count and the other had a low leukocyte count, and in only one of the cases were both the temperature and leukocyte count low. We have had cases with a temperature of 104° F. and a leukocyte count of 8000. In an article I published about three years ago on the diagnosis of appendicitis I insisted that the patient must have some elevation of temperature before a diagnosis of appendicitis should or could be made. I remember a case that occurred very early in my experience. A doctor called me to see his daughter, and he gave me a typical history of appendicitis—the local sensitiveness, pain, nausea and vomiting, but no elevation of temperature. He insisted that the child had no elevation of temperature. I took her to the Presbyterian Hospital and had her asleep on the table ready for operation when I decided to make another examination before making the incision. The absence of temperature elevation made me just a little suspicious that it was not a case of appendicitis. I made the examination, and in the palpation the tumor which I detected preceding the anesthesia and which was so sensitive suddenly slipped up under the costal arch. It was the kidney, and not the appendix, which was at fault. The kidney had been displaced. She had been to a picnic two days before, and while she was leaning up against a board fence, a young man lifted her up by her feet and rolled her on the top board of the fence. The top board just caught the kidney, and it was torn loose and pushed down into a subperitoneal position at the McBurney point. She did not tell this story to her father. But she did not have an attack like an ordinary kidney case. The condition cleared up in a short time. We did not get the correct history from her until after she had recovered from the anesthetic. It was a classic history of appendicitis, except for the absence of temperature elevation, and it was that that made me suspicious.

We insist on elevation of temperature at least some time during the attack. When there is no temperature elevation in the middle of the attack, it means that there is no continued absorption of toxic products from the appendix, but all these cases have a rise of temperature in the beginning. It may not be detected, but it is there just the same. In cases of streptococcic infection there is often a leukocyte count of 75,000 or 80,000. The streptococci run through the lymph-spaces of the appendix like water goes through a sieve. In the ordinary case of appendicitis, due to staphylococcus infection where pus is retained in the appendix, the organ is very sensitive, swollen, and red, but there is an absence of the tension which is present in the pneumococcus infections.

We have had some cases very closely allied to appendicitis in their clinical history which may well be mentioned at this time. One was a young man whose doctor called me up at 6 o'clock on Sunday morning to see him. I came over to the hospital and found the man in acute pain. He gave a history of having had a similar attack in the summer. At that time he was motoring down the Hudson River, when he was stricken with severe abdominal pain, nausea, and vomiting. He was so sick that he had to be taken into a farm-house and put to bed. In a few hours he recovered and was able to continue his journey. The next attack was the one he had when I saw him. The doctor had diagnosed it as appendicitis. I told him that no one ever got over an attack of acute appendicitis of the severity of the Hudson river trip attack in so short a time. On examination by our fist percussion method I was able to make a diagnosis of kidney stone. The urine was now examined and found to contain some red corpuscles. Five days later, while in the hospital, he passed the stone. Another man came in with very similar symptoms. I examined him by the fist percussion method, and, by the way, fist percussion is the best method to use in trying to make a diagnosis of stone in the kidney, and when I percussed him over his right kidney zone he screamed and said, "Doctor, it is all I can do to keep from striking you, you hurt me so."

I have been told that the appendix in this patient's father was situated very high up. I did not see the case. The father developed a subphrenic abscess, so the boy's mother says, and died.

I am going to make our usual incision through the aponeurosis of the right rectus, well on the outer edge but higher up than usual. We never cut the muscle; we simply displace the muscle-fibers inward. Then we go through the posterior sheath, pick up the peritoneum with two tissue forceps, and incise it on the incline so as not to injure the intestine if it happens to have been grasped by the forceps. Thin pus is coming out already. There is quite a lot of it. I think he has an extensive periappendicitis, if his story is correct.

The next thing is to get that appendix. Inserting my finger through this opening, and sweeping it over toward the right side, I come across a band of adhesions on the outer side of the caput coli, leading to the appendix. I will free these adhesions without making much traction on the intestines. There are some firm organic adhesions around this appendix, showing that he had had a previous attack. The appendix is behind the colon, and its distal end is way up near the liver. I must be very careful now because I do not want to rupture the appendix.

The patient said that he had pain in the right lumbar region in each attack. That corresponds with the findings because the appendix is way up in the right lumbar region and adherent beneath the ascending colon and to the posterior peritoneum, which accounts for his lumbar pain. I can just touch the tip of it with my finger. I have it free now. It is about four inches long and distinctly clubbed. There is no perforation. He had a streptococcic infection without a perforation. The appendix was located just where his father's was, but the father had a subphrenic abscess because ten days elapsed before he was operated on. This has been a very difficult operation—one that could not be done in five minutes. We will amputate the appendix in the usual manner. The mesentery of the appendix is ligated and then cut away from the appendix. Then we crush the appendix near the cecum, and ligate it in the crease where the mucosa and submucosa have been displaced. Then we put in the puckering string in the cecum around the base of the appendix, beginning the suture by making the over and over stitch of the mesentery. This stitch ligates the appendiceal artery and prevents hemorrhage from it later. That is really a Lembert stitch, made with Pagenstecher linen. We then amputate the appendix just above the ligature. It is very thick walled, and shows the evidence of recent attacks of inflammation. The stump of the appendix is cauterized with 95 per cent. phenol, and without being neutralized, it is inverted into the cecum, and the puckering string is drawn tight. A few additional Lembert sutures are put in over the inverted appendix, and that portion of the operation is completed.

We will put in a tube drain behind the cecum and outside of the peritoneum. A second smaller drain will extend down into the pelvis. That procedure is necessitated by the infection which was present in the peritoneal cavity.

Let the record show that a large drain was inserted extraperitoneally on the outer side of the ascending colon and caput coli; that a smaller drain goes down into the pelvis. The lower peritoneal drain is sutured to the larger upper drain and is not sutured to the skin.

Let the record show further that the appendix was fixed way up under the ascending colon, the tip directed upward toward the liver, and that there were many adhesions present, which made the operation a difficult one. There was a considerable quantity of seropurulent fluid in the peritoneal cavity, but the appendix had not ruptured either intraperitoneally or extraperitoneally. The appearance of the peritoneum was that of a streptococcus infection. It is red and glossy. We had to enlarge the opening up about 3½ inches in order to get at the appendix. We were not able to reach it through our original incision.

From the appearance of that appendix this morning I think that the man's statement that he did not have pain on pressure last night was absolutely wrong. He was bluffing us and bluffing himself. He had recurrent appendicitis. The enormous thickening of the wall of that appendix shows that. On opening it we have some difficulty in splitting it. It is firm and white. The mucosa is thickened and reddened.

The peritoneum will be closed with catgut, the cut edges being turned out, making an ectropion. The rectus aponeurosis is closed with catgut, and the skin with horsehair. Figure-of-8 silkworm-gut sutures are put in before the horsehair closes the skin. These sutures bring the wound edges closely together, so as to obliterate all dead spaces. The wound is dusted with bismuth subiodid, a desiccating powder, and the drainage-tubes are secured with a sterile safety-pin. Adhesive straps are placed across the incision to give support to the abdominal walls, and to prevent the wound being torn open by straining or retching, if there is any.

This man will be put in a sitting position and will be given 20 c.c. of streptococcic serum right away, and proctoclysis for twenty hours.

[The patient made an uneventful recovery, and left the hospital three and a half weeks after the operation. The drains were removed at the end of a week; the stitches after two weeks. —Ed.]

Proctoclysis Treatment of Dr. John B. Murphy

Dr. Richard L. Stoddard, Rochester, N. Y., furnishes this description to his nurses and to the patients, if necessary, to guide them in the administration of the proctoclysis treatment:

Therapeutic Indications.—In the various forms of peritonitis, post-operative or otherwise, and in general toxemic conditions, as in typhoid, scarlatina, also in the early stages of pneumonia, before the heart has suffered from the intoxication. In the later stages of pneumonia it should be used with great caution. Immediately after all severe operations with low blood-pressure or annoying thirst it is very useful.

The saline solution is made by adding one dram each of sodium chlorid and calcium chlorid to each pint of hot water. The solution must be kept at a temperature of 100° to 101° F.

The average quantity given is 1½ to 2 pints every two hours. The quantity to be given depends upon the severity of the case, the age of the patient, and the development of an edema. The average twenty-four-hour quantity is 18 pints, or 1½ pints every two hours. Less than 8 pints in twenty-four hours is of little value. In a child of eleven years as much as 30 pints were given in twenty-four hours.

The base of the saline solution container should be elevated sufficiently (2, 4, or 6 inches above buttocks of patient) to allow 1½ pints of the solution to flow into the rectum in from forty, fifty, or sixty minutes' time. The height of the container should always control the hydrostatic pressure. The solution should never have a hydrostatic pressure of more than 15 inches.

The rapidity of the flow should never be controlled by the use of forceps, clamps, or knots in the tube: a medium-size tube with several openings in the tip is to be used. Provision should always be made for a sudden return of the flow into the container when the patient strains during the act of coughing or vomiting, or wishes to expel gas or fluid. If this return is provided for, the patient rarely soils the bed.

The patient is placed in the Fowler position, and the saline treatment is continued for about three days, but rarely five or six days. When emesis is present after operation, gastric lavage is the best means of relief.

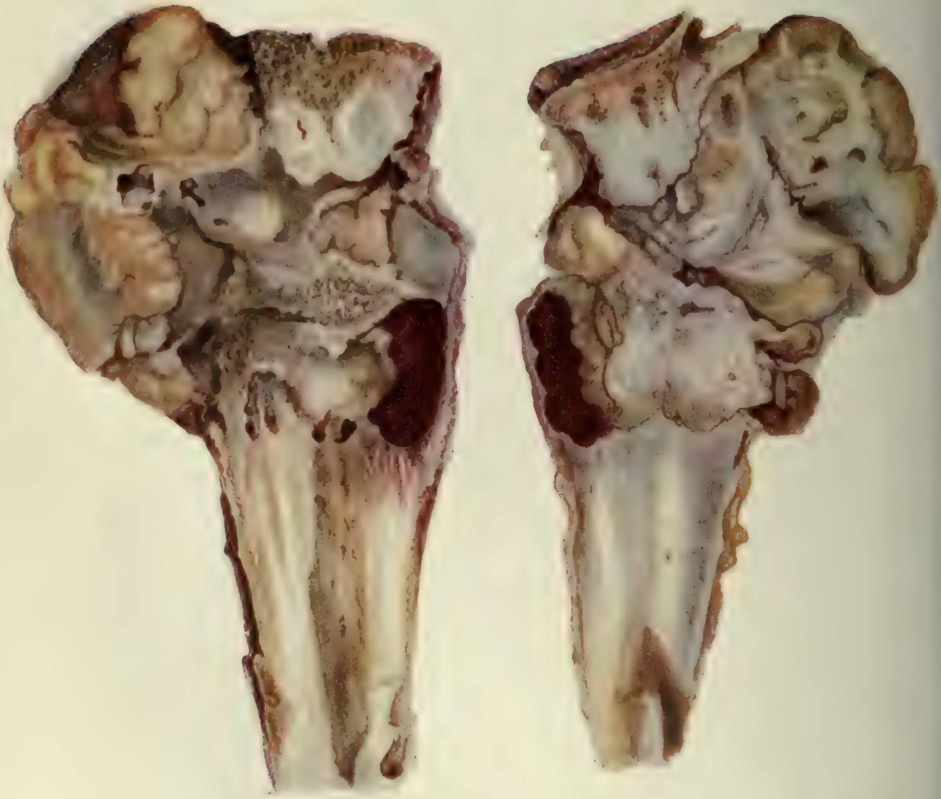
Too much solution after the third, fourth, or fifth day is indicated by an edema of ankles, hands, and even the face; the solution should then be discontinued until the circulatory equilibrium is restored, when the treatment may be repeated, if indicated.

A medium-size hard-rubber vaginal douche tube, flexed at an obtuse angle two or three inches from its tip, causes no inconvenience if so strapped to the thigh as not to press on the posterior wall of the rectum. Frequent changing, as removing and inserting, also an improper position of the tube or too rapid a flow of the solution into the rectum, are each and all very annoying to the patient, and soon produce an irritation of both the anus and rectum, resulting in a partial or complete evacuation of the saline solution. The tube may remain in twenty-four to forty-eight hours without change.

NOTES.—In case one of the elaborate and up-to-date solution containers is not at hand, I find that an ordinary douche-can may be used as a useful and satisfactory container for the saline solution, which may be maintained at the desired temperature by surrounding the container with bottles filled with boiling water, also immersing one or two bottles in the solution. To further retain the heat the whole apparatus (bottles and container) may be wrapped in a warm woolen blanket. I believe that the consensus of authoritative opinion is that a temperature of 105° F. to 110° F. is preferable to that of a lower temperature.

I have thoroughly tested the "*Murphy method*" of giving the proctoclysis treatment in peritonitis, typhoid, uremia, diphtheria, pneumonia, shock from hemorrhage, and local and general septicemia. I have found it of unquestionable value as a therapeutic agent, and especially so if used early and before the heart has been affected by the intoxication.

FRONTISPIECE



Sarcoma of upper end of tibia, not involving the articular surface. Just below the inner tuberosity the bone was eroded. The tumor is split open. A fragment of apparently normal bone may be seen on the upper edge.

OBTURATION ILEUS: OBSTRUCTION DUE TO LARGE GALL-STONE IN ILEUM

HISTORY

PATIENT, female, unmarried, aged fifty, enters hospital on account of pain in left lower quadrant of abdomen, continued vomiting, and weakness.

In July, 1912 (seven months ago), the patient was taken ill with severe pain in right upper quadrant of abdomen. This came on very suddenly and lasted one day. She began vomiting about one hour after onset of the pain, and the vomiting continued at intervals during the day. Vomitus was bile colored. Pain did not radiate to shoulder, and she was not jaundiced.

In October, 1912 (four months ago), she had a similar attack, with pain in the same region,—the right upper quadrant,—not definitely localized in any one place. The vomiting continued for nearly three days and was bile stained. She was not jaundiced.

At Christmas-time, 1912 (two months ago), after eating a heavy meal, she was taken ill with very severe pain in the abdomen, in the same region as in the previous attacks. This lasted for two days. No radiation of the pain. The vomiting started almost at once, and was quite severe. She was not jaundiced. Was very constipated in all these attacks, but is not sure whether there was a complete stoppage of the bowels. At no time was she ill enough to call in a doctor.

Tuesday, February 11, 1913 (ten days ago), she ate a heavy meal at noon. At 3 o'clock in the afternoon she had a severe pain over entire upper abdomen—much more severe than at any previous time. She did not go to bed, but took a walk, thinking it would help her. Between 6 and 7 o'clock in the evening she began to vomit, and the vomiting continued until the present time. Every two hours or so she becomes very nauseated and vomits bile-stained material. On Tuesday evening she noticed that it had a very offensive odor. She cannot keep anything in her stomach. Has not been able to have a bowel movement since onset of trouble, neither can she expel gas. Enemas return clear. She can retain no cathartics in stomach. Abdomen seems slightly distended. The last two days the pain seemed worse in the left lumbar and iliac regions.

Patient always has been very constipated. Never any mucous or bloody discharge. Thinks she had no fever in any of the attacks. Always been unusually well and strong, except for present trouble. Has lost no weight. Has had no night-sweats and never was jaundiced.

COMMENTS AND OPERATION

DR. MURPHY: This woman had recurrent attacks of vomiting with fecal retention. She has had no bowel movements since February 11 (ten days ago); nothing came through the bowels. She is tender to pressure in the left iliac fossa. She has been having salines since her admission into the hospital. Examination by the rectum is negative. She has had regurgitant vomiting for ten days. Some gas came through and the vomiting ceased, but the emesis returned yesterday. It is not an adynamic obstruction, and it is not a strangulation obstruction. It is an obturation obstruction.

We will enter the abdomen through the left rectus muscle. She has a very large abdomen, and we have to extend our incision high up. Nothing resembling a tumor could be felt at any time; no history of bloody or mucus discharges. There is a possibility of the obstruction being caused by a carcinoma, but that is negatived by the absence of blood and mucus in the stools.

Having gone through the skin, rectus muscle, fascia, and peritoneum, we are ready to make the examination. There is a dilatation of the small intestine and there is free fluid in the peritoneal cavity. It is not bloody. This dilated small intestine shows that the obstruction ought to be in the ileocecal region. There is no dilatation of the large intestine. The sigmoid is not dilated. The next thing is to palpate the transverse colon. The transverse colon is not dilated at all. That shows us that the obstruction is in the small intestine. Now I am under the great mesentery. There is the trouble. It is a huge gall-stone in the ileum that had ulcerated through the gall-bladder into the intestine. It is a faceted gall-stone, showing that it was one of many stones.

I once had a case of fecal concretion that had been in the bowel for twenty years. The man had eaten a mass of plum-pudding when a boy, and from that time until we operated he had recurrent attacks of pain. We opened the abdomen and found a mass in the small intestine which proved to be plum-pudding covered with calcareous material.

This was a very pretty case. It is pretty because it shows the length of time the stone remained quiescent. When it began to move, she got the colic. That is in accordance with the statement we made many years ago that it is the moving body that causes the colic. That stone has been arrested in the bowel for eighteen months.

We will incise the bowel over the stone opposite the mesentery and take out the calculus, exercising great care to prevent the field from becoming infected. Two rows of Pagenstecher linen and one row of mattress stitches of catgut will close the opening. The stone is disk-shaped, about one inch in diameter, and three-quarters of an inch thick. Its upper and lower surfaces are concave.

This patient gave no history of a jaundice, which shows that this stone escaped from the gall-bladder into the intestine by ulceration through the wall of the gall-bladder into the small intestine. This must have occurred about a year and a half ago, at the time she had the initial symptoms. If it had passed through the common duct into the duodenum, she would have had a severe colic and jaundice as well, which was never a symptom at any time in the course of this case. She gives a history of having had disturbances in the right hypochondrium some years ago.

The reason we did not make an exploration of the upper abdomen at this time was that she is in the eighth day of her obturation ileus. The diagnosis of obturation ileus was made before the operation, and it has been verified by the operative findings.

In locating that obstruction we followed a definite plan, which we have used for many years. First we expose the sigmoid, unless we see a contracted coil of small intestine in the field when the abdomen is opened. If one sees a contracted coil of small intestine, then the first point to seek is the ileocecal valve, because then we know that the obstruction is above the ileocecal valve. When the ileocecal valve is reached, the ileum is explored. If the obstruction is above that, it will show a contracted ileum. We then pass the small intestine through the fingers, keeping them all the time within the abdomen, so that nothing will be permitted to escape through the abdominal wound. When the obstruction is reached, it is removed through an opening in the bowel.

These obstructions occur much more frequently in the right iliac fossa than in the left. They occur where bands develop from the remnant of the omphalomesenteric duct attachment—that is, 39 inches above the ileocecal valve. It is the most common place for the formation of annular contractions of the intestinal wall that are dense and firm enough to produce an obstruction if the intestine passes beneath the band, but also the most common place for the formation of intraperitoneal bands, as well as the site for attachment of the remnant of the vitello-intestinal duct or Meckel's diverticulum. The bands must be elevated, and the point of their crossing carefully examined, to see that necrosis has not taken place from the strangulation at the junction of the bands. The band is then freed, and the strangulated colon brought up and its vitality determined; namely, whether it will live or not. If it is decided that it will live, then the omentum is wrapped around it and the bowel replaced in the abdomen. The omentum may be tacked to its mesentery. This is a safe thing to do in that class of cases, because when the omentum is wrapped properly around the bowel, it rapidly becomes adherent, and if necrosis does occur, perforation is prevented by the omentum. If there is only a small dark area, where the band has compressed the intestine, one-

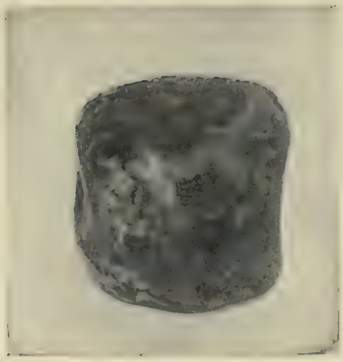


Fig. 67.—Obturation ileus. Full-size reproduction of the gallstone found in the ileum.

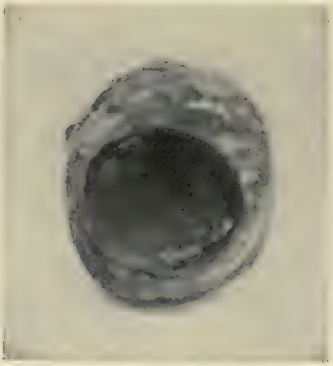


Fig. 68.—Obturation ileus. Under surface of the stone, showing one of the large and well-marked facets. There was a similar facet on the upper surface, showing that this was one of a number of stones.

fourth or one-eighth of an inch wide, this can be covered over by an annular Lembert stitch and an involuting stitch of catgut, which is often sufficient to protect that weakened field, and it does not produce intestinal obstruction subsequently. One small drain will be inserted here, and the abdomen closed in the usual manner.

The basis on which we made the diagnosis of obturation ileus was the fact that she had vomited and that repeated salines had relieved the severity of the attack; second, that this condition has been present for eight days without the patient going into collapse; third, in obturation ileus the patient may go three, five, ten, and even fifteen days without a bowel movement, and still show no effect of the fecal or general intoxication, while in strangulation ileus, when the vomiting once begins it continues to increase in severity to a fatal termination, if not relieved. Just before the fatal termination a brownish-black liquid with a distinctly fecal odor is vomited in a projectile way. The most serious of all symptoms of intestinal obstruction of the mechanic variety is the capillary cyanosis. Cyanosis occurs in the capillary circulation twelve to fifteen hours after the onset of the symptoms. The temperature is often subnormal. The pulse increases rather rapidly in frequency—out of all proportion to the increase in temperature and the leukocytosis. The leukocytosis is not common, but is sufficiently frequent that it cannot be used as a differential symptom between obstruction and infection. McDonald reported a 30,000 leukocyte count in an acute intestinal obstruction without gangrene or infection. In the obturation type some fluids probably pass by the obstruction and some of the patients expel gas, but no feces.

The obturation ileus may result from—

(*a*) Neoplasm.

(*b*) Cicatricial contraction of the lumen of the bowel without a strangulation of the circulation.

(*c*) Volvulus of at least three-fifths of the circumference of the sigmoid.

(*d*) From fixations of the bowel through adhesions from the wall of the abdomen, as occurs in postoperative ileus.

(*e*) From foreign bodies—(1) Gall-stones, the most common;

(2) fecal concretions; (3) neoplasms—fibrous adenoma of the intestine.

(*f*) Invagination of the intestine—intussusception. In these cases the intestine is on a border-line between strangulation and obturation, from a symptomatic as well as from a clinical standpoint.

(*g*) When the intestine is invaginated to a severe degree, the inner pouch or the intussusceptum is strangulated by the fixation of the intussuscipiens at its neck, this also causing an occlusion of the mesenteric vessels and bowel lumen at the point of entrance of the intussusceptum.

Emesis is a very important symptom in making the differential diagnosis between the obturation and the strangulation type of ileus. The fecal vomiting or the general vomiting of the small intestinal content is not at all uncommon. Pure fecal vomiting we have only seen twice in our entire experience, showing that fecal vomiting as a symptom of intestinal obstruction has practically no value, but vomiting of the small intestinal content is of value in diagnosis. Repeated washings of the stomach after small intestinal content vomiting do not regularly show a continuation of the obstruction. The disappearance of the colon material and the offensive odor from the stomach in the repeated washings show, in all probability, that the obstruction has ceased, which was so beautifully illustrated in this case forty-eight hours ago.

Of the notable symptoms in connection with intestinal obstruction, the vomiting of "fecal" matter is one of the danger-signs of impending disaster; second, the capillary cyanosis; third, the elevation of the pulse; fourth, the temperature is frequently subnormal.

In the management of these cases the first and all-important point when the physician is called is to empty the stomach by repeated lavage. Have the patient transferred to a position where the intestinal obstruction can be relieved at the earliest moment. If it is the obturation type, one must be prepared to do very extensive operative work. Resections of the small intestine are easy to make, end to end, with the round button, and for this class of cases, in the hands of the experienced surgeon, this should be the method of election. In making the resection, a sufficient portion of the colon above the point of obstruction should be resected so as to remove the cyanotic, necrotized, infiltrated band of intestine, and get into the zone of the normal intestine. The removal of two or three feet or more of the intestine is of no moment in the final result. The treatment of intestinal obstruction was considered by me in an article written some three years ago. The salient points in it are as follows:

In the specific relations of choleliths to intestinal obstruction we can well analyze the pathologic lesions on the following basis:

1. The stones that pass through the wall of the gall-bladder into some portion of the intestinal tract.

2. The stones that pass through the wall of the cystic duct into the intestinal tract.

3. The stones that pass from the common duct by ulceration through its wall into the intestinal tract.

4. Stones that pass from the gall-bladder through the cystic and common ducts into the duodenum.

The rôle of gall-stones in the intestinal tract represents the superlative degree of pathologic changes in the gall-stone cycle. The points at which the gall-stones produce obstruction in the intestinal tract can well be judged from Courvoisier's analysis of 52 cases, and we believe this represents the average position of arrest of the stones in the intestinal tract:

Duodenum	3
Jejunum	4
Upper jejunum	1
	8
Upper ileum	4
Middle ileum	1
Ileum	18
Lower ileum	10
	33
Ileocecal valve	7
Vermiform appendix	2
	9
Sigmoid flexure	2
	52

These statistics of Courvoisier are of especial value, as they show the clinical course in the cases preceding the era of frequent operations for gall-stone disease. They correspond fairly well with those of LeConte, Gibson, Lichtenstern, Moynihan, Woods, Porter, and Murphy.

The positions of obstruction are very interesting. A lady, eighty years of age, had 22 stones in a diverticulum of the duodenum, which produced all sorts of digestive disturbances. In one patient there was an obstruction of the ileum by two stones not larger than a hazelnut at the site of a malignant stricture. In two cases there were perforations of the appendix from impacted gall-stones. In two others there were large conglomerate masses of choleliths in the intestine. In a case reported by Bermond there was a mass of stones as large as two fists, and in one by Puyroyer a mass as large as one fist. Of 125 cases, 70, after various and repeated colics, emesis, peritonitis, ileus, etc., were cured spontaneously by the passage of the stones per anum. From the analysis of the cases it was impossible to tell with any degree of certainty how the majority of the stones came into the intestinal tract, as in those cured spontaneously it was not ascertained, and in many of those on which a postmortem was made it was not recorded. In 36 cases in which the report was accurately given the stones were passed into the intestine through a gall-bladder-duodenal fistula, in 25 cases a gall-bladder-ileum fistula, in one a colon fistula, in one a duodenal and colon fistula, and in seven a choledochus and duodenal fistula.

The size of the stones that can pass into the common duct by the wedge-shaped dilatation may well be estimated from an analysis of these cases: In one, where the patient died after five days of symptoms of ileus, the stone measured four inches in circumference; the choledochus had contracted down after its passage to the size of the index-finger. In another the patient died after fourteen days of icterus from a cholesterin stone impacted in the ileum. In one a herniotomy was performed for an incarcerated hernia, but without symptoms of obstruction in that zone, and it was found that there was an enormous gall-stone filling the intestinal lumen. The choledochus was as large as the stone. In one case there was an obstruction with two large cholesterin stones at the ileocecal valve. In this case there was no evidence of perforation of the surface of the gall-bladder, but the duct was still large. Another patient who had had an arrest of half of a large gall-stone died suddenly a few days later, and it was found that the other half had become impacted in the intestinal canal, ulcerated through its wall, and caused a perforative peritonitis. The choledochus was dilated to the size of the gall-bladder. In still another instance the patient died on the seventeenth day of the ileus from diffuse suppurative peritonitis due to an ulceration of the gall-stone through the tract. In one instance a lady aged sixty-seven years suffered four days from ileus. Laparotomy was performed; two large gall-stones were removed from the small intestine, but she died in twenty-four hours from the septic peritonitis.

It will be noted in all these cases that the ileus terminated fatally a long time after the onset of the symptoms of intestinal obstruction. That is due to the fact that the patients were suffering from an *obturation ileus;* that the stones were constantly changing their position, and, therefore, did not perforate, and, further, that with obturation ileus the patient survives a much longer period of time than with strangulation ileus, notwithstanding that in both there is a complete retention of the material above the point of obturation.

The intestinal obstruction by the stone itself may be analyzed as follows: The ileus due to gall-stone which has perforated through the gall-bladder into the intestine may have no preceding jaundice, but the inflammatory symptoms which accompany such a perforation ought to suggest the diagnosis. There may have been colicky pains preceding the perforation by the stone while it was impacted in the neck of the gall-bladder, but there is no colicky pain while the stone is in the fundus of the gall-bladder itself. If an ileus develops after an attack of colic or a prolonged colicky jaundice in women of middle age, the possibility of gall-stones should be considered. If the stone perforates into the colon, it causes few, if any, symptoms, while when it perforates into the small intestine, the characteristic attacks of ileus are produced, and these may be repeated for months or years, depending on the advancement of the stone or arrest in diverticula in the intestinal tract. It may result finally in the escape of the stone into the large intestine, its ulceration through the wall of the intestine, with perforation, peritonitis, etc. The arrests are more common a few inches above the ileocecal valve than in the valve itself, as in the former position the lumen of the small intestine is less than at the ileocecal valve.

Intestinal obstruction may be defined as a complete or partial obstruction to the onward movement of the bowel contents, which obstruction is of *mechanical* origin. *Ileus* includes the stasis of the intestinal contents from excessive muscular contraction of the wall; *dynamic*, or paralysis of the peristaltic wave from any cause; *adynamic*, or *mechanical* obstacles to the advancement of the fecal current.

Obstruction may be unassociated with strangulation, and this is the common condition in gall-stone ileus, in intestinal polyp, enterolith, etc. Strangulation ileus involves not only the suspension of transmission of the intestinal contents, but a suspension or retardation of the circulation in the bowel segment. Strangulation ileus is a much more rapidly fatal condition than obturation ileus. In a single case of gall-stones we may have the three varieties of ileus:

1. The adynamic variety, while the stone is passing through the neck of the gall-bladder, cystic duct, and common duct.

2. The dynamic variety, where a small stone produces a spasm of the intestinal wall, with all the symptoms of ileus, and where the stone is much smaller than the lumen of the intestine, as reported by Courvoisier.

3. The mechanical variety, where the stone is large enough to occlude the lumen of the bowel completely and arrest the fecal current.

The clinical history will show symptoms of preëxisting cholecystic disturbances, either colics and cholecystitis, or cholecystitis, colics, and jaundice. These may have existed for a number of years preceding the intestinal obstruction. The intestine may be obstructed through adhesions or flexions of the intestinal tract produced by paracholecystitis. Volvulus and incarcerations may also occur as a result of gall-stones. The symptoms of the mechanical occlusion of the intestine are:

Pain, always of a colicky character. The spasm or colicky waves decrease in frequency after the first twenty-four hours. They may be reëxcited after the muscle-wall has become fatigued by long efforts at expulsion of the foreign body; by kneading or percussing the abdominal wall. The abdomen is not very sensitive to slow pressure in the early stage.

The appearance of the *vomitus* when the stone is high up is of the gastric and biliary type. When lower down, it may take on the fecal color, but it rarely ever has the typical fecal odor. The vomiting increases in frequency as the hours of the attack increase, until the stone either ceases to advance, by becoming arrested in a diverticulum, or escapes to a place where the intestinal content can pass by it. The quantity of emesis is less the nearer the stone to the pylorus. The great bulk of the material is made up of the transudation into the intestinal tract above the point of obstruction. It is not due to reversed peristalsis, but to the accumulated products of secretion and alimentation above the obstructing body. With the contraction of the intestinal wall the material cannot advance downward, and must find its escape at the upper and only outlet. It, therefore, is forced back into the stomach and is ejected. In the beginning it is associated with nausea. As the case advances vomiting becomes projectile; later it may become a mere gulp, and flood over the patient's entire face. The vomitus is rarely ever fecal, that is, having the distinct odor of feces.

Borborygmus (peristalsis) is of the periodic type. It usually begins some distance from the point of obstruction, increases as it reaches the point of obstruction, and terminates in a loud explosion. The explosive point indicates the zone of obstruction. The stethoscope gives very valuable information in intestinal obstruction. A dose of morphin suspends the peristaltic waves from six to eight hours, and thus destroys one of the best aids to diagnosis.

Tympany may be local or general: local, and in the upper abdominal region, when the duodenum or upper jejunum is the site of obstruction; general, when the calculus is in the ileum or at the ileocecal valve. Circumscribed areas of flatness can frequently be discerned. They are due to the accumulations of fluid above the point of obstruction. Their relations to definite obstructions were outlined by von Zoege-Manteufel. Recurrent attacks of obstruction in these cases often develop great dilatation of the bowel above the obstructed zone.

Coprostasis forms the key-stone of the arch of symptoms of intestinal obstruction. In obturation ileus small quantities of feces may be passed from time to time and even a little gas. Both are misleading symptoms, and lure the physician on in the hope that the ileus is of some other than the mechanical type, until perforation or other dangerous complications occur. The possibility of erroneously interpreting these signs should be borne in mind.

With mechanical obstruction of the intestine there is practically never an *elevation of temperature* in the first forty to sixty hours. Later, when gangrene and perforation have taken place, elevation of temperature may appear. This absence of temperature elevation is very valuable in differentiating mechanic obstruction from the infective lesions within the abdominal cavity.

Leukocytosis is, as a rule, only moderately increased. We have seen, however, a 36,000 leukocyte count in a case of mechanical ileus.

Differential diagnosis will have to be made from, first, gallstone obstruction of the neck of the gall-bladder or the cystic or common ducts; second, from acute pancreatitis, infective, hemorrhagic, and fat necrosis. This is the most difficult condition to differentiate. The elevation of temperature, however, with the great sensitiveness to pressure under the costal arch and perpendicular percussion, and the early collapse are valuable aids; third, from tabetic crises; fourth, from perforative peritonitis; fifth, from appendicitis; sixth, from acute renal lesions: (*a*) metastatic infarcts (hematogenous infection); (*b*) obstructions in the pelvis; (*c*) obstruction to the ureters. These acute kidney lesions give evidence of great pain on heavy fist percussion over the diseased kidney. The presence of blood in the urine microscopically will aid in the differentiation. Seventh, embolism of the mesenteric artery or thrombosis of the portal vein gives a train of symptoms so closely resembling those of acute mechanical obstruction that preoperative differential diagnosis is rarely made; eighth, torsion of a tumor pedicle gives the pronounced symptoms of ileus, but the presence of the tumor aids one in the diagnosis; ninth, fecal impaction may be of the recurrent obturation type, and only through the recognition of the fecal mass by palpation or proctoscopy with megasigmoid or megacolon can it be differentiated.

As many of the conditions mentioned above are surgical lesions, demanding immediate surgical treatment, the importance of differentiation is not so great so far as the life of the patient is concerned. These patients should be operated on at the earliest possible moment, before there is time for perforations, periintestinal adhesions, intra-abdominal abscesses, or acute intestinal obstruction collapse, a condition from which the patient rarely ever rallies. Delay in operating for intestinal obstruction is more fatal than delay in any other abdominal lesion, except acute gastro-intestinal perforations into the free peritoneal cavity. A free gastric lavage should be given immediately before operation. Local or general anesthesia may be used—both sparingly.

The incision should be made through either the right or the left rectus muscle, over the point of greatest sensitiveness and the location of the peristaltic explosion.

When the abdomen is opened, if all the presenting coils are greatly distended, the hand should be passed into the pelvis to grasp the collapsed ileum as near the ileocecal valve as possible. This should be passed rapidly through the fingers in an upward direction, the assistant returning the coils into the peritoneal cavity as rapidly as they are palpated. When the dilated fixed point is reached, great care should be exercised not to tear the intestine where its wall is weakened by necrosis or ulceration from pressure by the calculus. If the wall is in fairly good condition, it should be divided transversely and the calculus removed. The intestine should be closed by a double Lembert suture, thus turning into the lumen of the bowel the fold that was most damaged by pressure from the calculus.

In looking for points of obstruction, when the abdomen is opened there are dangers of missing the obstructions in the periduodenal subperitoneal pockets, diaphragmatic hernia, pericecal pockets, and retrosigmoidal peritoneal diverticula. When the nutrition of the intestinal wall has been materially interfered with, a resection of the bowel should be made, and a considerable sized segment of the bowel removed. Many postoperative perforations have been due to the fact that too little of the infiltrated and damaged bowel above the point of obstruction was excised.

In the small intestine the end-to-end operation is the one of choice, but when the wall is damaged, side-to-side or end-to-side, by suture or oblong button, gives the best results. Immediately after the laparotomy, if the patient is materially depressed or collapsed, an intravenous injection of 1.5 to 2 pints of salt solution should be administered before he leaves the table.

Many intestinal cases die cured of the obstruction, *i. e.*, the collapse continues to a fatal termination a few hours after the operation. The exact cause of this collapse is unknown. It never occurs with patients operated on within a few hours after the onset of symptoms, not even when a large segment of the intestine is resected. Cardiac, nerve, or vasomotor stimulants, with the possible exception of intravenous injections of adrenalin, have little or no effect in these cases. Drainage is rarely ever indicated. Proctoclysis should be instituted as soon as the patient is returned to bed, and a pint and a half of normal salt solution administered every two hours for twenty-four to forty-eight hours. Six to eight ounces of solution is of little or no value, and the practice is not to be recommended. Anodynes are rarely ever indicated after operation. Heroin is the best to use when one is necessary.

The clinical history not infrequently entirely disarms one, from a diagnostic standpoint, as these large stones ulcerate through the wall of the gall-bladder into the small or large intestine, and therefore the patient may never have obstruction symptoms, as when the stone escapes from the gall-bladder through the cystic and common ducts. Enormous stones can pass through the common duct without producing obstruction symptoms, as the following case indicates.

One of these large stones was encountered in the common duct of a doctor from Vancouver. It was one of the largest stones I have observed. It measured 1⅞ inches in length and 1⅛ inches in diameter. It came into the duct without a colic, and was well on its way to the ampulla of Vater at the time it was removed. Its enormous size prevented the spasm of the duct—the colic that is usually present when small stones are passed. This case is one of the exceptions to our law that "jaundice of any considerable duration, not preceded or accompanied by a colic, is not a gall-stone jaundice."

The absence of jaundice is an indication of the fact that the stone is not coming through the gall-bladder, the common duct, the cystic duct, or the hepatic duct.

The prognosis in this class of cases is very good.

Another fact of importance is the presence of colic and its relation to all cases of this class. In our article on "Intestinal Obstruction and Cholangic Infections," written eighteen years ago, we said:

"Colic is the manifestation of the advancement or advancing of a foreign body in the lumen of a canal or duct. When the foreign body becomes stationary, the colic ceases, and the pain and colic may be absent for months or years. When the foreign body starts to advance from its diverticulum, either in the gall-bladder, the ureter, or in the intestine, there is a recurrence of the symptom of pain. That foreign bodies can remain embedded for years in the ureter, the common duct, the cystic duct, or the intestine is illustrated by cases too numerous to mention. I recall one which occurred in my serivce in the Cook County Hospital, and which is mentioned in my article on 'Intestinal Obstruction' in Keen's 'Surgery'—the case where the patient had taken a large quantity of plum-pudding when he was a boy, cited above."

The cessation of the obstruction symptoms, in this case forty-eight hours ago, was indicative of the arrest of the body. The recurrence of the pain and emesis showed that the stone was again progressing, although the diameter of the stone showed that it was sufficiently large to produce an obstruction of the ileum. It might, however, have lodged higher up—about 39 inches above the ileocecal valve, where it first becomes arrested, as this is the contracted portion of the intestine, and occasionally it has a diverticulum. The ileum then expands and remains expanded up to a point three inches above the ileocecal valve, where it again contracts, and this is the narrowest point in the entire small intestine. It is a law that foreign bodies produce ulceration below the ileocecal valve, rather than above it.

[The patient made an uneventful recovery. The drain was removed on the fifth day, the stitches on the twelfth day.—Ed.]

INTESTINAL STASIS CAUSED BY BAND OF ADHESIONS

HISTORY

Patient, male, aged twenty-seven, enters hospital on account of abdominal pain and very severe constipation. He has always been subject to constipation. Present trouble dates back two years, when he noticed a sharp, severe pain in his rectum, radiating up into his pelvis. This has continued off and on till the present time. When bowels do not move for several days, the pain becomes more severe. At the present time the severe pain persists for three or four days, and then disappears for five days. With this pain comes great difficulty in securing bowel movements. Cathartics seemed to have no effect, and when given an enema, it is often retained. He does not seem to be able to expel it. Has frequent desire to defecate, but often cannot evacuate the bowel. Has had a mucous discharge during the past two years. Occasionally, during the past year, he has noticed slight traces of blood—last time was three weeks ago. At present the stools are flat and ribbon-shaped. Has lost 15 pounds in weight during the last two years. Has drenching night-sweats at irregular intervals. They last a week or ten days, and then disappear for a week or so. Has no cough. Is very nervous, and recently seems to tire very easily. The only way he can secure a bowel movement now is by an enema. Cathartics seem to have no effect.

COMMENTS AND OPERATION

Dr. Murphy: This is not a tumor of the intestine—it is an intestinal stasis. Did you say he had rectal pain?

Intern: Yes.

Dr. Murphy: He did not tell me that. He said he suffered from severe constipation. He has gotten along with cathartics of increasing strength, only he has reached the limit. The statement of his pain is not the one he gave to me. He has some bloody discharge, a condition that comes with the chronic constipation accompanying tuberculosis of the intestines. There is no history of fever. The slimy mucous discharges were the sequence of a chronic colitis. We did not estimate the condition as being one of intestinal tumor. From the statements read by the intern one would be suspicious of an intestinal tumor, but there is no tumor palpable. There is nothing to make me think it is a tumor; no history of tuberculosis, the only suggestive symptom being a discharge of this quantity of blood. If it is a tuberculosis, it is higher up in the abdomen than usual.

The condition of the patient is this: he is out of commission. This is one of the cases that comes under the classification made by Mr. Lane. He is out of commission because of his autointoxication and his constipation. It is not only in his bowel now—it is in his "head."

Examination of the rectum shows absolutely nothing visible or palpable from below. We are, therefore, probably going to short circuit the large intestine and attach the ileum to the sigmoid. We will do that operation if the conditions indicate it. There is nothing in the history to indicate the presence of a colitis that would result in a pericolitis; no dysentery. He has no evidence of an enlarged colon. He never passes large masses of feces. We did not make the high proctoscopic examination.

The results in these cases, as interpreted by Mr. Lane,—and he is the man whom we all follow in this work,—have been rather striking. He advocates the removal of the entire large intestine, which he did in a number of cases, and got a good result as far as the relief of the symptoms is concerned. More recently he has modified his position and does not resect the entire large intestine in all cases; he short circuits the large intestine, and attaches the ileum to the sigmoid, closing up the space between the mesoileum and the mesosigmoid, so as to prevent the falling of the small intestine through the loop. If we do that operation, we will make an end-to-side union of the ileum to the sigmoid. We will probably do that with the round button in place of the suture, but we will put the suture outside of it, just the same as we do a gastro-enterostomy. This operation should not be done without careful consideration of the patient.

This man has sought relief at many clinics. He has been advised to have a short-circuiting operation done, and finally he has arrived here for operation. Now, then, if we can find some pathologic condition other than a simple retention or stasis, we will be mighty happy. We like that better than to have to do a short-circuiting operation for a stasis. It was believed by Mr. Lane that the removal of the large intestine would be the operation of election in these cases. He has had little backing up of the feces into the large intestine from the short-circuiting operation. It seems to me that I read an article where that occurred and where a subsequent operation had to be done. I cannot recall the article at this time, but I feel quite certain it was not from Mr. Lane's pen.

Short-circuiting of the intestine for tuberculous lesions is a very old operation. We have been utilizing for twenty years now the exclusion operation of the Germans; that is, where the tuberculosis involves the caput coli, the transverse colon, a large portion of the descending colon, or any of these portions where there is an infiltration of the mesentery and where there were organic adhesions. It was used because of the fact that when you short-circuited the intestine for tuberculosis you threw the tubercular ulcer out of the fecal current, and the tendency is for it to repair. Some years ago Dr. L. L. McArthur operated on a young lady for tuberculous stenosis of the bowel. He did the short-circuiting operation. She also had a tuberculosis of the intestines, and he put her on the tuberculin treatment. He made an end-to-side union with the button, and she has been perfectly well since, not showing the slightest evidence of a continuation of the tuberculous process. This is quite in contrast with Virchow's early belief. He said he never saw a case of tuberculous ulcer of the bowel that had been fully repaired preceding the advent of tuberculin. She is almost as broad as she is long. Repair of tubercular ulcers has been reported any number of times since tuberculin has been used. Preceding the use of tuberculin, in the Pathologic Institute at Berlin, Virchow reported that the tuberculous ulcer would not repair. Paul Guttmann reports a number of cases of ulcers of the intestines in patients who died of tuberculosis of the lungs, and who had been given continuous treatment with tuberculin. As it was given them in very large doses, all the bowel ulcers were healed. That was the first time Virchow said he ever saw a healed tubercular ulcer, showing how rare repair was at that time.

I am going into this abdomen through the right rectus muscle, close to the midline. We go through the skin, fat, fascia, and rectus sheath, displacing the fibers of the rectus muscle outward, so as not to have the division of the aponeurosis on the same plane as that of the muscle-fibers. The peritoneum is then opened in the usual manner. There is the sigmoid. We draw it up into the field. There is no tumor of the sigmoid, no thickening of its walls, and no dilatation. Now we will palpate the large intestine, and as we near the liver, we find the cause of the obstruction. It is a firm band of adhesions coming from the duodenum, passing clear around the transverse colon to the back, and out on the opposite side. There is where the obstruction occurred. He has no history of a pericolitis or pericholecystitis, and still that is where the duodenal fixation, which Mr. Lane has described, occurred. He has an extensive perisigmoiditis. That is indeed a pathologic condition.

I will cut and ligate that band. The intestine is now perfectly free. I will cover all of this raw surface with omentum. The adhesion band which exists over the sigmoid as the result of a perisigmoiditis is large and firm, about 2½ inches broad at its base, and covered about 6 inches of the sigmoid; the sigmoid was plicated over on itself in the pelvis. A short-circuiting operation is not indicated now. We merely divide and ligate all the adhesions.

Now you can see why the enema was not returned. That band was the cause of his trouble.

We will examine his appendix. It is embedded in the wall of the caput coli. It is distinctly pathologic. It is very long, looped, and completely embedded by adhesions. The tip is directed upward. There is considerable increase in the vascularity of the caput coli. In handling this case one cannot get away from the conviction that there were many conditions leading to this obturation condition.

Let the record show that the appendix was about 6½ inches long. It was a continuous source of irritation, judging by the adhesions to the caput coli. It was embedded in the wall of the caput coli as the result of inflammatory adhesions.

This was a very interesting case. I am convinced that he is going to be relieved by the operation.

Let the record show that there was no shortening of the meso-ileum and no evidences of tuberculosis—no tumor; that there was no dilatation of the colon, meaning that there has been no organic and continuous obstruction, merely an obturation obstruction from these adhesions.

We close the abdomen, layer by layer, everting the cut edges of the peritoneum. Catgut is used for the peritoneal and rectus aponeurosis sutures; silkworm-gut for the figure-of-8 tension sutures, and horsehair for the skin. The usual dressing is applied.

[This patient got along very well. He had a normal bowel movement on the third day after the operation. His bowels continued to empty themselves naturally once and twice a day afterward. The patient left the hospital four weeks after the operation.—Ed.]

PARATRACHEAL TUMOR—CYSTIC ADENOMA OF THYROID

HISTORY

PATIENT, male, aged forty, enters hospital on account of severe hoarseness and smothering and choking spells at night, and because of a tumor in his neck which he says is on his "windpipe." On the morning of December 18th (two months ago) he awoke and found himself very hoarse. He was able to speak only a word or two, and then he could not get out a tone above a whisper. The night before he had coughed a great deal, and he was awakened many times. He had had a cold for about a month previous to this time, accompanied by a mild cough and coryza. His throat was sore for one week. His voice cleared up slightly during the morning in question, but at night it became worse again, and he was unable to speak above a whisper. He had a very severe cough from the onset of the hoarseness—an irritating dry cough. He felt as though he had considerable phlegm in his throat, but he could not raise it. His cough came in spells, and would be so severe as nearly to convulse him. This condition continued for nearly ten days. He was practically unable to speak during this time. Then his cough became much worse, keeping him awake all night, and following a spell of coughing he would complain of severe pain in the left lower side of the chest and occasionally in the right side, up near the axilla. Also following the coughing he would seem to have a "lump in his throat." He felt suffocated and struggled for breath. The slightest exertion would precipitate a spell. Often he was wakened by one at night. He felt better when he was up. Lying down seemed to suffocate him. The condition remained about the same until his entrance to the hospital.

Six weeks ago he began to have nosebleed. It bled first on one side and then on the other. The bleeding usually occurred in the morning. He still has the nosebleed. When his coughing spells became severe he had a burning sensation down in his throat. Then he vomited a green liquid. This vomiting only occurred after a severe coughing spell, however, and bore no relation to food or feeding. Seventeen days ago he noticed that he was at times passing bright-red blood from his bowels. For two weeks the stools were tinged with blood. He had no chill at the beginning of the trouble, and thinks he had no fever. Had no night-sweats. Had pleuropneumonia six years ago. No other serious illness. Has had a slight, hacking cough all his life, but no expectoration. Venereal history negative.

COMMENTS AND OPERATION

Dr. Murphy: That case is a very odd one. His symptoms came on suddenly, the first being the coughing. How much does he drink?

Intern: He says he drinks quite a little beer, but not much whisky.

Dr. Murphy: He looks like a man who might have a typical alcoholic hoarseness—a whisky hoarseness. But this hoarseness came on suddenly; then he got better again. He had another spell, which continued for a week or ten days. Then he began to have nosebleed and hemorrhages from his bowels, and they continued for two weeks. That bleeding has stopped now, and he is here with a small mass on the side of the trachea. We had the laryngologist examine this. He says there is no involvement of the vocal cords, and that the hoarseness is due to pressure on the larynx.

The mass here was felt in the throat near the midline. It does not pulsate, therefore it is not an aneurysm. Neither is it an abscess. There is no fluctuation present. It is apparently attached to or is a part of the thyroid. It is not an enlarged thyroid in the real sense. It feels like an encapsulated mass, separate and distinct from the thyroid. There is no evidence of an enlargement in the larynx, or indication of an existing infection outside the larynx, which so frequently occurs with the tubercu-

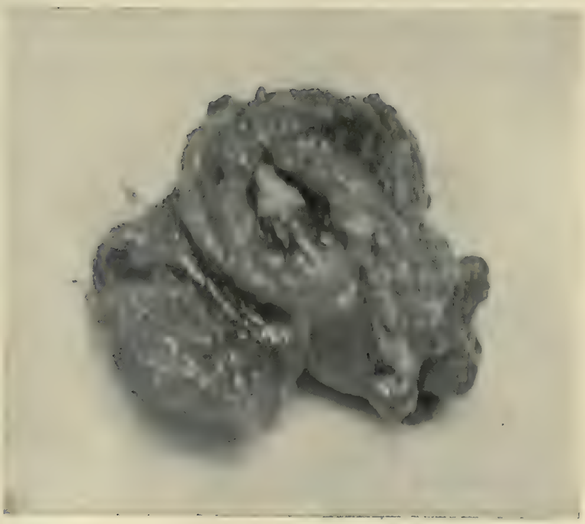


Fig. 69.—Paratracheal tumor. Cyst of the thyroid laid open, showing thick capsule and débris in cyst.

lous variety of laryngitis or the carcinomatous variety of laryngitis. He has the type of cough that would result from an acute irritation of the recurrent laryngeal nerve. Another lesion to be considered here is hematoma. Is this a hematoma that has formed in the paratracheal tissue and that is pressing on his recurrent laryngeal nerve? I have asked Dr. Tivnen to look him over, and, after a careful examination, he has come to the conclusion that the mass is outside of the trachea, and that it is pressing on the larynx, causing his cough and hoarseness.

I am going to expose this mass in the same manner that I would remove a tumor of the thyroid. A transverse collar incision will be made, extending through skin, fascia, and platysma. I am going to divide a portion of the sternocleidomastoid on this side, so as to get well around this tumor, and examine it and outline it before I do anything else. I will divide the sternohyoid group of muscles. There is no evidence here of an enlarged thymus. By retracting the sternocleidomastoid the tumor, for such it appears to be, comes into view. It is pressing on the trachea. It looks like a thyroid neoplasm. Whether it is malignant or non-malignant will have to be determined later. It does not pulsate, and it does not look like a hematoma It feels hard and firm, but scarcely firm enough to be malignant. It is encapsulated. It apparently originated on the wall of the trachea, and compressed the recurrent laryngeal nerve, giving rise to the train of symptoms described. I can enucleate the tumor very easily. There is practically no hemorrhage.

The wound will be closed in the usual manner. The divided muscles will be reunited with catgut, and the skin with horsehair.

Tumors and cysts in this position frequently compromise respiration. Many years ago a colleague of mine had a cyst as large as a goose-egg in this position. He persisted in not having anything done for it. After an acute cold, he suddenly got an attack of asphyxia. He was in the neighborhood of the College of Physicians and Surgeons, and the late Dr. Alexander Hugh Ferguson punctured the tumor with a trocar and saved the man's life. The tumor proved to be a cyst. In another case, in 1884, I was called to see a young lady who was deeply cyanosed and almost pulseless. She had a small tumor on the right lobe of her thyroid, which was compressed between that and the clavicle and had flattened and caused absorption of the trachea. Division of the isthmus of the thyroid permitted of elevation of the tumor out of its confined position and she was immediately relieved.

[The tumor proved to be a cystic adenoma. The patient made an uneventful recovery.—Ed.]

DESMOID TUMOR OF THE RECTUS MUSCLE

HISTORY

Patient, female, married, aged thirty-one, enters hospital on account of tumor in right rectus muscle above umbilicus.

Patient first noticed this tumor six weeks ago, when she was having some breast trouble. She had a smarting sensation in region of tumor. On palpation she discovered a lump, quite hard and fairly movable. Six months ago, in the beginning of the last three months of her last pregnancy, she suffered a more or less constant pain in this location, as she describes it, like something pinching or pulling at her skin of the abdomen. It got worse as the pregnancy advanced, and disappeared after the baby was born. Two years ago, during the last three months of her first pregnancy, she experienced the same sensation in this region, and it too disappeared after the birth of the child, but at neither time did she discover any mass there. Has not noticed any change in size, shape, or consistency of the tumor during the past six weeks. It is never tender.

Past history is negative. Always well and strong. Menstrual history normal. Has had two pregnancies, the last completed about seven weeks ago. Mother, father, and brother died of carcinoma.

COMMENTS AND OPERATION

Dr. Murphy: This is a very rare case. I have not seen more than a half-dozen of these cases, showing what a rare type of tumor it is. I think I have seen five. This is the third to come to operation.

There is a very strong family history of carcinoma here, but the patient is a young woman. She just had her second child. During the last three months of gestation she complained of some irritation in the upper abdomen. It continued until after confinement. When you palpate this tumor, first it appears to be in the abdomen; but when she takes a full breath, the tumor seems to be thrown outside of the abdominal wall. That is a very important sign in connection with desmoids, and to which we called attention some years ago. Ordinarily, the tumor appears to be a part of the muscle.

The term desmoid is used to apply to very hard, fibrous tumors or fibromata of the abdominal wall. It is not a pathologic term at all—it is purely a clinical term. These tumors are rather uncommon. They occur most frequently in young women between twenty and thirty-five who have borne children, although they have also been observed in the nullipara and even in men. In the greater proportion of the cases the tumor is first noticed by the patient some time during pregnancy, and they have been known to take on a very rapid growth during that time. We do not know of any reason for the association of these tumors with pregnancy.

Their usual location is in the posterior layer of the rectus sheath, above the umbilicus, more often on the right side than on the left. They have been observed less rarely in the abdominal wall, attached to the costal cartilages and the crest of the ilium. It has been supposed by some that rupture of the rectus muscle during labor may be the etiologic factor in the production of these tumors, and the fact that the muscle usually ruptures from its posterior surface would lend some color to the correctness of this belief. However, it must be remembered that these tumors grow from the sheath and not from the muscle itself, therefore rupture of the muscle would not be a direct, but merely an indirect, factor in the causation of desmoids in this locality. They are always fibromata originating in the connective tissue of the sheath, and not in the connective tissue in the muscle.

In shape the tumor is often ovoid, rarely ever being spheric, globular, or disk-shaped. The size of these tumors varies from that of a pea, weighing a fraction of an ounce, to a mass weighing many ounces. The tumor is not definitely encapsulated. It infiltrates the tissue in which it is found, so that its attachment is

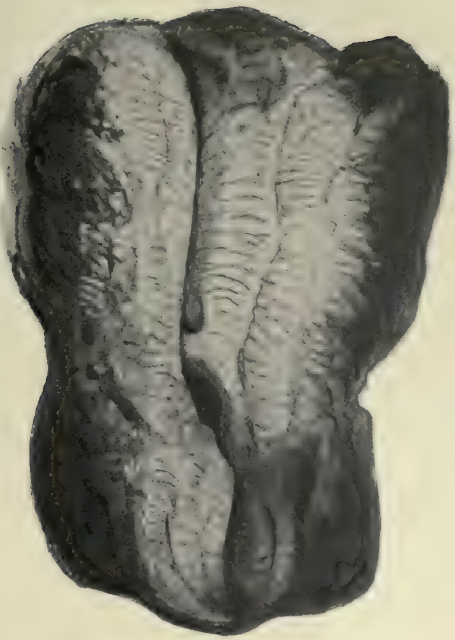


Fig. 70.—Desmoid tumor split longitudinally, showing the density of the fibrous structure.

rather firm and there is an ingrowing of connective tissue into the tumor, as well as an outgrowing from the muscle into the connective tissue. The tumor may undergo calcareous infiltration or myxomatous degeneration.

Microscopically, the tumor is a pure cellular fibroma. The borders of the neoplasm are infiltrated with muscle-cells. These tumors are usually of slow growth, and, therefore, the symptoms to which they give rise vary both in number and in intensity. Usually the patient is inconvenienced only by the size of the growth, when it is large, although sometimes it is very painful, and if the mass projects into the abdominal cavity, additional symptoms may be caused by the formation of intraperitoneal adhesions. When situated below the umbilicus, they may be mistaken for pelvic tumors, and cases are on record where, the mass being situated above the umbilicus, a diagnosis of carcinoma of the stomach was made.

When these tumors are removed, there is but little tendency to recurrence, although recurrences have been reported. Opinion varies somewhat on this point. Stone, for instance, in 1908, analyzed the clinical statistics of these tumors and said at that time that recurrence was noted in 90 per cent. of the female and 68 per cent. of the male patients. Pfeiffer, on the other hand, who wrote on this subject quite extensively some time before Stone's paper appeared, estimated the recurrences in women at only 21 per cent. In the 5 cases we have seen there has been no evidence of recurrence after removal of the growth.

The treatment consists in excision of the tumor without opening the peritoneal cavity, if it can be avoided. The size of the mass will determine the extent of the operation. In large tumors it may be necessary to open the peritoneal cavity in order to make a complete excision. We always endeavor to preserve the anterior sheath of the muscle in order to prevent the occurrence of a ventral hernia.

We will make a large incision directly over the mass, incising the skin above and splitting the anterior sheath of the rectus and exposing the neoplasm. You will note that the tumor is very hard, because the scalpel grates as I cut into the mass. We will not remove any more of the rectus muscle with the tumor than is necessary, so we will go wide of the margin,—about one-half inch,—so as to make sure that we have enucleated all the tumor-cells. We are going to do all we can to prevent a recurrence.

The tumor is about the size of my index-finger. When I palpated it before the operation through the abdominal wall it appeared to be about as large as my fist. It occupied the right rectus muscle, just to the right of the midline, and just above the umbilicus. We succeeded in enucleating it without opening the peritoneal cavity, which is rather gratifying. The anterior sheath of the muscle was not involved, so that we will be able to give this abdominal wall a firm support by suturing the anterior rectus sheath very carefully with strong catgut, imbricating the margins of the incision.

On section of this tumor you will notice the hard white tissue. It looks very much like a sarcoma, and that is one of the tumors from which desmoids must be differentiated. It is really a type of fibrous sarcoma or cellular fibroma, with a very low degree of malignancy. We do not know whether these cases ever metastasize clinically or microscopically. I will endeavor to draw together the edges of the rectus muscle, shortening it, so that this abdominal wall will have a good support.

Let the record show that this tumor involved three-fourths of the diameter of the right rectus muscle, just above the umbilicus—that is, about one inch; that we were able to separate it from the peritoneum without opening the latter; that it originated from the posterior rectus sheath, growing outward into the rectus muscle.

The abdominal wall is closed with a figure-of-8 and horsehair sutures. The patient will be advised to wear an abdominal support for a long time, so as to give these tissues every opportunity to unite firmly and solidly.

[Microscopic section of this tumor confirmed the clinical diagnosis of desmoid. The patient made an uneventful recovery from the operation. The stitches were removed on the fourteenth day, and the patient left the hospital on the twenty-second day feeling entirely well.—Ed.]

PLASTIC OPERATION ON EAR

Ear Bitten Off by a Horse

HISTORY

Patient, male, aged forty, enters hospital on account of injury of the right ear. January 27th a horse bit off about two-thirds of the external ear. It bled profusely. The ear was bandaged. No sutures were used. Hearing is perfect. Operation February 3, 1913.

COMMENTS AND OPERATION

Dr. Murphy: The reconstruction of the ear is the most difficult plastic operation—that is, to make anything like a substitute for the ear. This ear is in very bad shape, but we are going to try to rebuild it. This man has a pyocyaneus infection on the base of the ear. He will be given a pyocyaneus vaccine right away.

We have planned to reconstruct this ear by taking one flap from in front of the ear, over the region of the zygoma, to make the top of the ear, and a second flap from behind, over the mastoid region, to rebuild the middle third of the ear. The top flap will contain a portion of the zygoma with its attachment at the ear, so as to give support to the top of the new lobe. This flap will be tongue-shaped, and it will be doubled on itself, so that a piece of the zygoma about one inch in length will be in the middle of the flap. In that way connective tissue will be approximated with connective tissue and the flap will be covered by skin above and below. The bone in it will make it stand up, and then the free edge of the flap will be sutured to its base. The denuded areas from which these skin-flaps are removed will be closed with horsehair sutures.

A large tongue-shaped flap will be removed from behind the ear, over the mastoid region, the base of the flap being directed toward and just on the edge of the upper ear margin. This flap will also be doubled on itself in the middle, and its edges will be sutured to the flap above and to the remaining portion of the ear below. The anterior flap is about 3 inches in length and the posterior flap about 2½ inches in length.

That will make a very good-looking ear, and without any very great degree of mutilation of the face. All the sutures will be made with horsehair, with as little tension on them as possible. One suture will be passed through the middle flap so as to approximate the anterior and posterior parts of the flap, thus permitting their union and preventing the formation of a hematoma.

The entire field of operation is then freely dusted with bismuth subiodid powder. A wire cage properly fashioned is then placed over the ear, the edges of the cage being well padded with cotton. The cage is held in place with wide adhesive strips. A roller bandage is then applied to hold in place a dry dressing put on over the cage.

[The subsequent progress of this patient was a very satisfactory one, the wound healing primarily. The stitches were removed on the seventeenth day. The pyocyaneus infection, which was dreaded at the time of the operation, yielded very quickly to the vaccine treatment. Nothing untoward happened. When the patient left the hospital, about four weeks after the operation, he had a very good-looking ear. When a second operation is performed on him for the purpose of straightening up the tip of the ear, bringing it a little closer to the head, the ear will be nearly as good-looking as its fellow. The result to the patient was a most gratifying one.—Ed.]

Second operation May 12th: The drooping which remained after the primary operation was corrected by sliding the base of the new-formed ear upward on the squamous portion of the temporal bone, and bringing the zygomatic process in the flap to right angles with the skull, thus acting as a tent-pole to sustain the elevation of the ear. There was no evidence of absorption of the zygoma. This is the first time, to my knowledge, that the zygoma has been utilized for the reconstruction of the ear. It is fulfiling this purpose admirably.

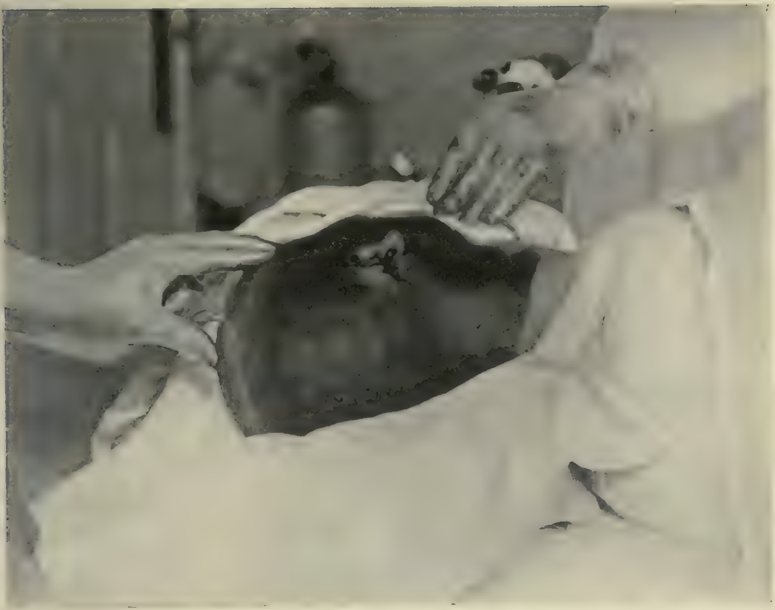


Fig. 71.—Ear bitten off by a horse. Photograph taken just before operation. All the ear except the tragus and antitragus bitten off.

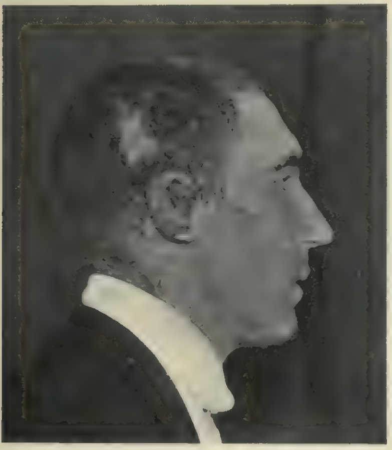


Fig. 72.—Lateral view. Six weeks after operation.

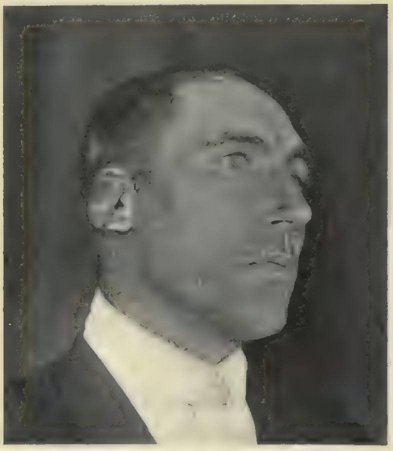


Fig. 73.—Anterior view. Six weeks after operation. The plastic flaps show very distinctly in this picture. There is still some swelling of the tissues.

TENOPLASTY OF FLEXOR TENDONS OF FINGERS

HISTORY

Patient, male, aged twenty-eight, enters hospital on account of inability to extend fingers of left hand and extreme flexion of fingers when hand is extended; also inability to supinate hand.

Present trouble dates back nineteen years. When patient was eight years old he fell from a second-story window and landed on his extended arms. Immediately afterward he had complete loss of control of both arms. He sustained a fracture of both arms, the fracture of the left arm being compound. Fractures were reduced and plaster casts applied, and both arms held in flexed position. The casts extended from fingers to shoulder, and remained on for several weeks. When they were removed, both arms were fixed in a flexed position. Forced extension was applied every day for several weeks, and the arms gradually extended. Does not remember ever having any disturbance of sensation in either hand. Patient also noticed, after removal of casts, that he could not extend fingers of left hand and he was unable to supinate right arm. Since then he has been unable to extend fingers when hand is extended, the fingers being firmly flexed; but when the hand is flexed, he is able to extend his fingers.

COMMENTS AND OPERATION

Dr. Murphy: Here is a case that is a surgical curiosity. This case also shows how misleading the skiagram can be as to the functional results. Skiagrams are being used very much in court, to the detriment of the physician, because the layman interprets the skiagram as to the functional results of a member after it has been fractured. The skiagram is no criterion as to the usefulness of a member after an injury. I am bringing in this case to accentuate that point.

When his hand is fully flexed, he can fully extend his fingers, but the moment his hand is extended he has a most complete and forceful closure of the fingers and thumb, indicating that the flexor tendons are too short. It is impossible to extend the fingers and thumb when the hand is extended. What about the swelling of his hand?

INTERN: It began swelling immediately after the bandage was put on.

DR. MURPHY: One of the indications that a bandage is too tight is swelling of the hand. This man is not in here because of the sequence of a fracture, but because of a sequence of too tight a bandage.

Looking at the skiagram, there does not seem to be much deformity of the wrist-joint. There is a little projection behind. The external condyle of the humerus is displaced three-fourths of an inch above the normal level. He can bring the arm up. He can put the forearm through the full range of motion, showing how much a joint can be deformed and still leave a serviceable arm. That is what I want to bring out. Often you see a small deviation and no function at all. Note the great displacement and the non-union of the external condyle in this case. The same type of fracture occurred in the other arm, but with a great displacement forward of the condyle. There is a non-union also there of the external condyle.

Still both forearms can be fully flexed and extended. There is a non-union of both external condyles of the humerus. There are perfect extension and perfect flexion with that frightful looking *x*-ray result. He has pronation and supination, but his pronation and supination are not in the right place. He has a good range of motion at the elbow, but it is not a normal joint so far as the radius and external condyles are concerned. It is a false joint. He cannot extend his fingers. That means that here we do not have to deal with either of the carpal flexors. His flexor carpi ulnaris and his flexor carpi radialis are all right. They do not have to be lengthened. What we have to deal with

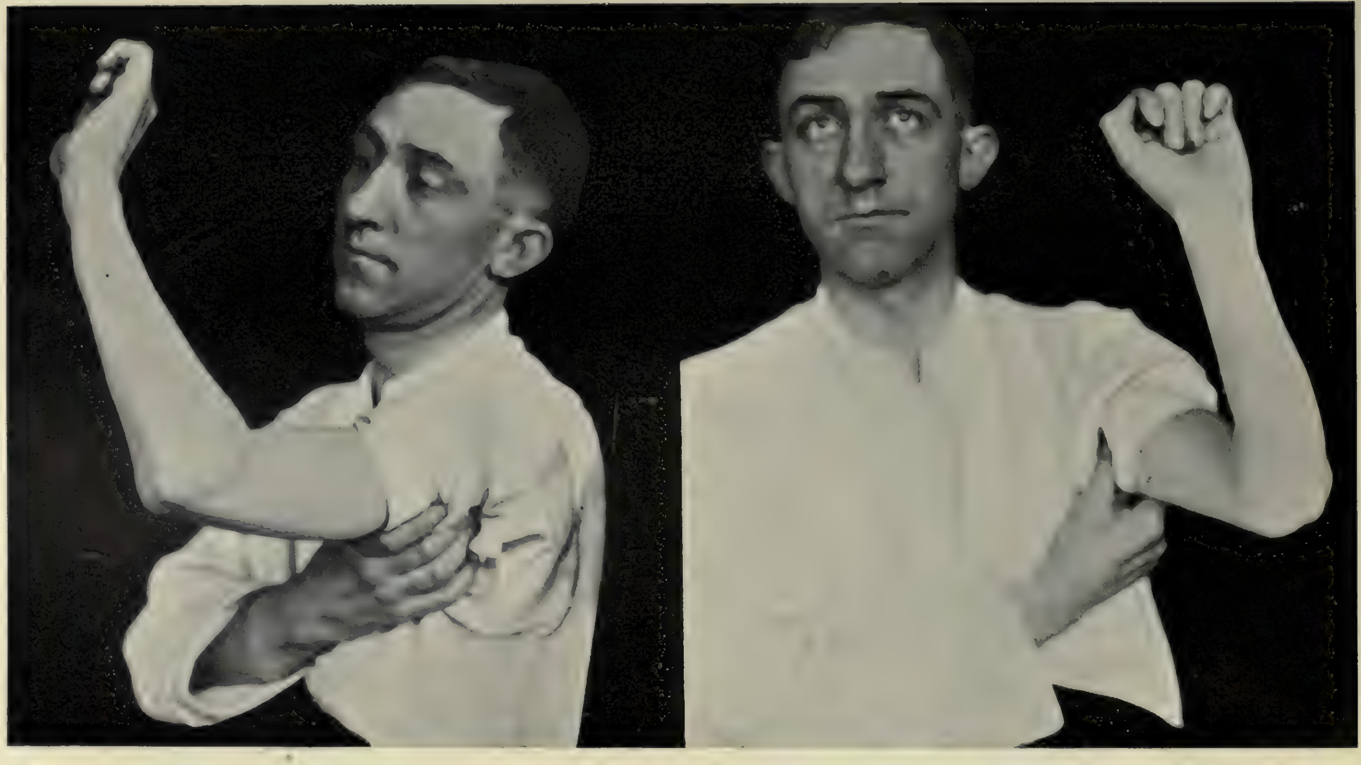


Fig. 74.—Tenoplasty of fingers. Side and front view, showing the contraction of the fingers at first joint, with extension at the second and third joints.

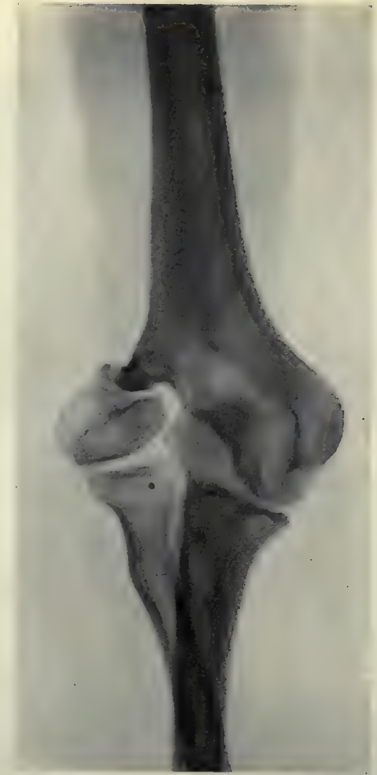


Fig. 75.—Anteroposterior view of elbow-joint, showing fracture of the external condyle, with dislocation upward of the head of the radius and separation of condyle from shaft. The radius articulates with the external condyle.

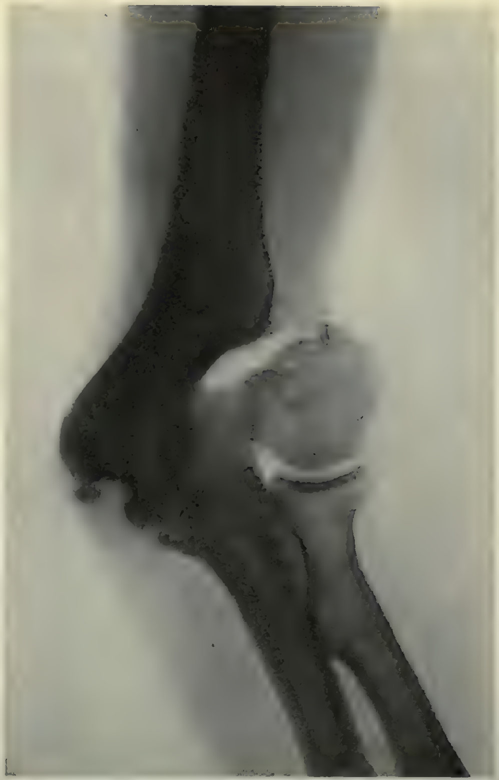


Fig. 76.—Lateral view, showing the marked dislocation forward of the external condyle. The articulation of the joint is otherwise normal.

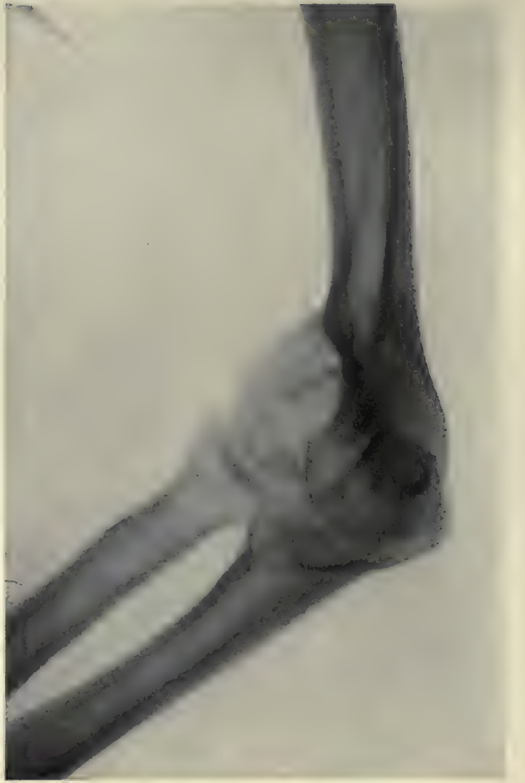


Fig. 77.—Another lateral view from the opposite side, again showing the position of the fragments and articulation of the bones, which gave patient practically normal elbow motion.

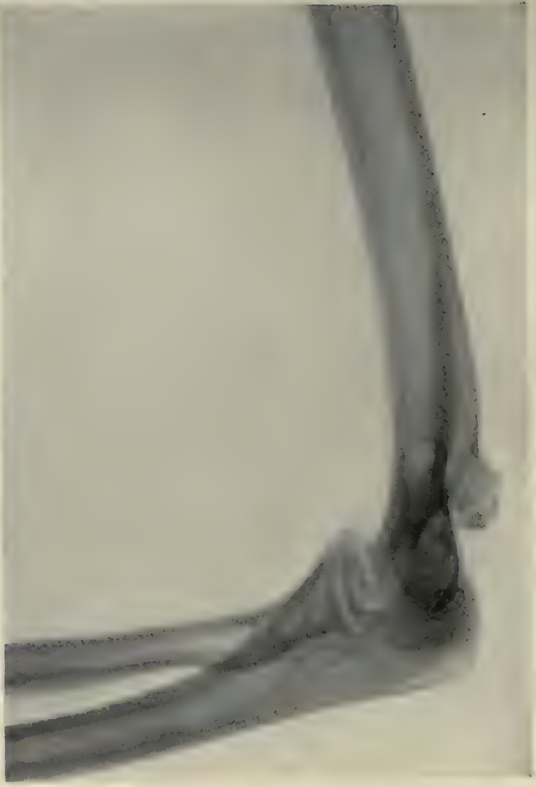


Fig. 78.—Lateral view, showing the degree of voluntary flexion in the joint. The external condyle fragment is not discernible.

is the long flexors of the fingers. There is no distinct nerve lesion.

We have had between 25 and 30 cases of Volkmann's paralysis. The muscles in his forearm feel like a bundle of violin strings, because all the muscle-cells are gone. All the mischief in these cases occurs within the first forty-eight hours. The tight bandage produces a traumatic myositis sometimes within twenty-four hours. We have seen one case where the injury was present as early as six hours after the application of the bandage. Muscular contraction from trauma of the elbow-joint is most commonly the sequence of too tight bandaging. This is particularly noticeable in the lesion described in the literature as Volkmann's contracture, Volkmann's paralysis, ischemic paralysis, and ischemic muscular atrophy with paralysis. None of these names, however, expresses the real pathologic condition, which is a traumatic myositis, the result of pressure produced by a circular bandage or splint applied too firmly against the muscles of the arm or forearm. Following such pressure there is an immediate myositis, with absorption and subsequent contraction of the muscle cicatrix. All the damage is done practically inside of the first seventy-two hours. The contraction shows itself in from four to six weeks, and assumes a fixed condition within ten to twelve weeks. It then remains stationary, except for the additional deformity which is produced by the growth of bone without a growth of muscle. This makes it appear as though the muscles were continuing to contract, which is not my view of the case.

There is no paralysis of the motor nerves from the ischemia, except when a splint presses against a nerve—the median, musculospiral, or ulnar. If one examines these patients carefully at the end of six or eight weeks, one finds that all the muscles respond to voluntary impulses; that the motion in them is limited by the fact that a greater or lesser portion of the belly of the muscle has been destroyed by the myositis, and now forms a firm cicatricial mass, often readily recognizable by a depression in the forearm or over the biceps or triceps.

When a bandage is applied too tightly around the knees, the external popliteal nerve is readily compressed as it passes over the outer surface of the fibula, just below the head. This results not in a muscular contraction, as it does everywhere else in the body, but in a paralysis and elongation of the muscle, which in the lower extremity is recognized as a drop-foot. It is a common sequence of a prolonged Buck's extension or a plaster cast applied firmly around the knee, or of compression in splints. It results in a paralysis of all the extensor group of muscles on the anterior surface of the leg, and later a contraction of the calf muscles follows, giving the patient a talipes equinus. The drop-foot so commonly present after fractures of the leg and thigh is a dressing sequence and not an injury sequence.

Occasionally, in traumas to the arm, forearm, thigh, and leg, the muscle bellies are cut through to the bone and extensive hemorrhage into them takes place, and the muscles become adherent or united to the periosteum, which fixes and immobilizes the muscle so that voluntary contraction or extension cannot be produced below the point of muscular fixation. Absorption of the adipose tissue may follow this contraction, and the skin is frequently attached directly to the aponeurosis or belly of the muscle. A myositis ossificans, when it extends into the muscle for a considerable distance, not infrequently produces a contraction and a limitation of motion. Intermuscular and intramuscular angiomata, the result of trauma or infection, may cause a phlebitis, which in turn causes a deposit throughout the muscle or muscle group of an extremity, resulting in contraction and fixation, with permanent deformity. A bandage that has been so tightly applied following an injury as to cause a rapid edema of the extremity below the point of injury or to cause sufficient pain to require an anodyne should be removed at once. At once means *immediately*—not in a few hours or in a few days. One must bear in mind that the pressure can destroy the muscle, as demonstrated experimentally, as early as three hours following the application of the bandage, and twenty-four to forty-eight hours is ample time in which to disable a member permanently.

Bandages and splints should never be applied for the purpose of reducing a fracture, and rarely ever for the maintenance of position by force after reduction. They are simply immobilizers or means for limiting voluntary or mechanical disturbance of the bones after reduction is effected. I have had under my care in the last three years 25 or 30 cases of this type, involving the upper extremity, which shows how common the lesion is. In not one of these was there a paralysis of sensation or motion, showing that the nerves were properly functionating. The limitation of motion was due to the contraction of the muscle following the pressure myositis.

The principles of treatment consist: (*a*) In the correction of the deformity; (*b*) in balancing the muscle groups so that subsequent contraction and deformity do not occur; (*c*) in the division of such portion of the joint-capsule as is necessary to permit of a full latitude of motion in the joint.

The first is accomplished by the elongation of the flexor tendons by means of a double L incision and division. The tendon should be elongated so as to leave a slack at the time of primary correction. If possible, parallel tendons should not be divided on the same line. Each tendon should have its sutures inserted at the time of division, but not tied. In the profundus sublimis pollicis group at the wrist the degree of elongation can be estimated by straightening the fingers and extending the wrist to the fullest extent after division and before approximation.

The flexor carpi radialis and ulnaris do not need to be elongated more than ½ to ¾ inch in any of the cases, while the index and middle finger tendons may need an elongation of 2½ to 3 inches. When the tendons have all been elongated and sutured, if the hand still does not come into a fully extended position, the capsule of the joint should be divided all along the anterior surface of the joint, and the line of division covered by a flap of tissue taken from the side of the wrist. When this cannot be accomplished, the point of union or approximation should be surrounded by egg-membrane to prevent tendon agglutination. When the sutures are tied and the tendons replaced, a portion of the tendon-sheaths or the fascia and fat from the inner or outer side of the arm should be swung in between the tendon groups and sutured, otherwise the tendons become fixed to each other transversely and the muscles move as a group in place of individually.

The division of the joint-capsule, associated with elongation of the tendons, does not receive sufficient attention in the literature on the subject. It is necessary to secure a permanent and a good result. After prolonged contractions in the neighborhood of the elbow, when the biceps tendon has been elongated, the forearm will not come down unless the anterior capsule of the joint is divided. Then it can be forced down gently, stretching the fibers of the brachialis anticus and the nerves, arteries, and veins in the trochlear space. Occasionally this stretching of the musculospiral and median nerves in front and the ulnar nerve behind produces a complete paralysis of the muscles of the forearm. This is due to the suspension of transmission of nerve impulses in the axons by what I believe to be an edema following stretching. It disappears in from two weeks to four months. In every case under my observation the function of the muscles was completely recovered.

If the anterior or posterior group of tendons are contracted and fixed to the bone to such a degree that a plastic operation cannot be performed, then an excision of the bone should be made, the forearm or arm shortened, and the muscles and tendons placed under favorable conditions for mobility, with muscular contraction and relaxation. Fat and fascia should be interposed between the tendons and the bone. If this cannot be obtained from the neighborhood of the injury, it may be obtained from the fascia lata of the thigh. It is essential that the periosteum and the skin should not become attached to the line of the new union of muscles or tendons, as these fixations are a great impairment to motion in subsequent muscular contractions and relaxation.

We will make a longitudinal incision on the anterior surface of the wrist and expose these tendons. We must make at least 2¾ inches of an elongation of the sublime flexors. We have made more than that much of an elongation.

It is peculiar how these tendons will reunite. If you direct them toward each other, it is wonderful the enormous distance they will span and reunite. The tendo Achillis will span the greatest distance and reunite. No other tissues in the body can be separated as much as this and reunite.

I am trying to save as much as I can of these tendon-sheaths. He has still his lumbricales and his interossei intact, both of which are short muscles and both of which act as flexors and extensors. I want to divide these tendons into two groups, so that these tendons will not become adherent in the same group. We do not have to disturb the carpal flexors. Each tendon is placed in a compartment by itself. The more compartments you can make or these tendons now, the less likely you are to have trouble subsequently from lateral adhesions. We keep these tendons from uniting to the skin at the line of union of the tendon by putting in subcuticular sutures of catgut.

The hand and fingers are in an extended position. There is no tension on the nerves at all. The skin is closed with horsehair. An anterior splint will be put on, leaving the tips of the fingers exposed. The patient will be encouraged to move his fingers slightly after two weeks, increasing motion gradually. In five or six weeks, after the tendons have united, active motion of the fingers will be required to prevent the occurrence of adhesions of the tendons.

[This patient left the hospital five weeks after the operation with the wound healed. He had good use of his fingers. He returned for examination on several occasions later. When seen last, normal motion in his fingers was almost restored.—Ed.]

ANKYLOSIS OF THE JAW

INTERPOSITION OF MUCOUS MEMBRANE FLAPS TAKEN FROM PALATE AND FLOOR OF MOUTH

HISTORY

PATIENT, a male, aged twenty-eight, enters hospital on account of inability to open mouth.

In July, 1909, he had an abscessed molar tooth in the right upper jaw, which was quite painful and which became swollen. He did nothing for three weeks; then the doctor opened the abscess from within the mouth and cauterized it. A week later the whole right side of his face was very much swollen. An external incision was then made to reach the abscess, but they did not seem to get much pus. Four days after this operation he noticed that there seemed to be some obstruction, other than that caused by the pain, which prevented him from keeping his jaws separated. This condition has been getting steadily worse. In December, 1912, he was scarcely able to open his mouth at all; something seemed to be holding his jaws together. He says the mandibular articulations were never sore or swollen. He had no distinct chill at any time. About one year after the onset of the trouble (August, 1910) he had an operation performed within his mouth to relieve the ankylosis, but it proved a failure.

COMMENTS AND OPERATION

DR. MURPHY: This man has a complete fixation of his lower jaw. He is unable to open his mouth, and on attempting to pry it open it is seen that his jaw is thoroughly ankylosed and that there is absolutely no motion.

In considering the ankyloses of the jaw we must remember that there are three types of ankylosis in this locality. There is, first, the fibrous type, which involves the joint itself, the articular surface being held in a fixed position by the formation of fibrous tissue; second, there is the bony type of ankylosis in which there is an ossific union between the head of the mandible and the glenoid fossa of the temporal bone. The articular cartilage has disappeared, and new formed bone has taken its place, and the mandible is solidly united to the temporal bone, and sometimes to the zygoma by the new-formed bone; third, there is the type of ankylosis the result of cicatricial fixation outside of the joint, such as may follow a large ulcer of the cheek, as is sometimes seen in typhoid or scarlet fever, following an abscessed tooth, or in an infection of any kind that is followed by the formation of a cicatrix, noma, etc. This type of fixation locks the jaw. When the fixation is relieved, the jaw drops.

It is evident that these varieties of ankylosis call for different modes of treatment. The bony type must be dealt with by an arthroplasty; that is, the bones must be separated at the line of union, and reunion is prevented by the interposition of a soft tissue flap made from the temporal fascia, as was done in this clinic recently. (See case of Ankylosis of Jaw in December, 1912, Clinics.) In the intra-articular fibrous ankylosis the same treatment is called for, because the mere division of the fibrous tissue, while it would cure the ankylosis for the time being, would not result in a permanent cure; therefore the interposition flap is demanded in this type of ankylosis, just the same as in the bony type. The fibrous tissue would redevelop, and in a greater degree than before. The fixation type of ankylosis is relieved more easily. Sometimes the mere division of the muscular attachments to the jaw relieves the fixation; that is, the attachment of the temporal muscle to the mandible may be divided; likewise the attachment of the pterygoid and masseter muscle, and in the case mentioned above the patient had such an operation performed on one side of the jaw, a typical arthroplasty having been done on the other side.

In the case before us today the ankylosis is of the extreme articular fibrous type. Cicatricial tissue is holding his jaw fixed. Our plan of procedure will depend entirely on the condition present; that is, how much cicatricial tissue there is and how much

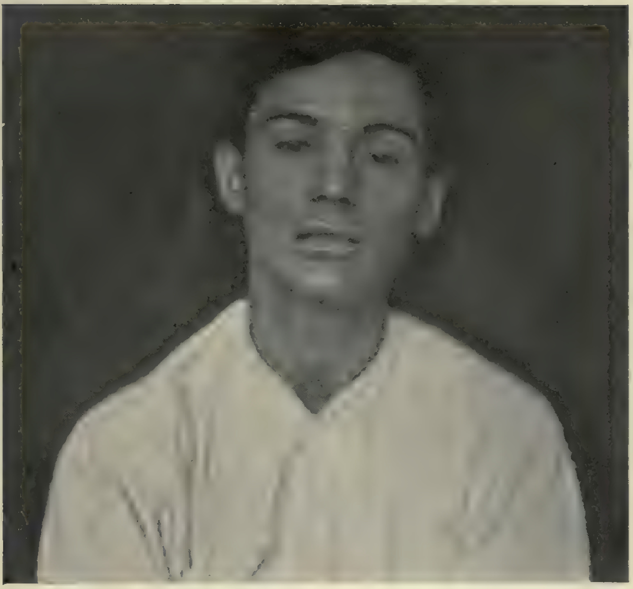


Fig. 79.—Ankylosis of jaw. Patient was unable to separate teeth farther than is shown in the picture—hardly an eighth of an inch.

mucous membrane is available for interposition between the ends of the divided cicatricial mass when we have separated the jaws. If it is possible, we propose to take our interposing flaps from the cheek; if not, we will resort to a new method, which we tried in a previous case, taking the mucous membrane covering the palate and the floor of the mouth.

On introducing my finger into this man's mouth, I find that there is an absolute fixation of his jaw. I cannot move it even a hundredth part of an inch. I can feel a solid fibrous band extending from the upper to the lower jaw, on the outer side, which easily accounts for the absolute fixation present. In order to give us more room it will be necessary to extract the only remaining molar tooth this man has in his upper jaw on this side. The cicatricial tissue I find extends clear back to the ramus of the jaw. The anesthetic in this case will be continued through the nose, and the mouth will be held open with a mouth-gag. I will separate the adhesions very carefully with the scalpel and scissors, using my finger as a guide. We will place a small gauze sponge in the back of the patient's mouth, so that the blood will not flow down into his trachea. We always have the tracheotomy tube ready in this class of cases. Sister and I had one experience with a blood-clot going down into the trachea to the bifurcation. We felt that we were going to have a death on the table. I immediately did a tracheotomy and tried to get out the clot. I failed in that attempt. Then I inserted a soft-rubber catheter with its end cut off into the trachea and sucked the clot out, much to our relief. The patient came out of it very nicely. That is one of the experiences that certainly teaches one a lesson.

We will try to wedge open this mouth as we go along with the division of the fibrous tissue. These cicatrices are very firm, and by prying the mouth open, the adhesions are put on tension, so that we can divide them very easily without endangering the other tissues. We have now succeeded in getting this mouth wide open. It took a long time and a lot of hard work, but we accomplished what we were after without an accident. In order to prevent the recurrence of the ankylosis by the reformation of the fibrous tissue I will interpose two flaps—one from the floor of the mouth and the other from the hard palate. By drawing the tongue over to the opposite side these areas are exposed freely; however, it will not be difficult to remove the flaps. Both flaps will be tongue-shaped, the lower one about two inches in length and one-half inch in width. The upper flap will be of about the same length but one inch in width. The base of the flap is directed toward the alveolar process in the case of the upper flap, and toward the root of the tongue in the case of the lower flap. Both these flaps will be swung outward to cover over the raw surface where the adhesions were divided. The tips of the flaps are sutured to the inner margin of the gum and the cheek. The denuded areas left after the removal of the flaps will heal over in a very short time. By using these mucous membrane flaps in the manner described the procedure in these cases is very greatly simplified, and it is not necessary to resort to the free fascia flap transplant.

It is absolutely necessary to keep these raw surfaces from growing together again. If we do not interpose flaps, this work will have been done for nothing. There really has not been a very great deal of hemorrhage. What there was was easily stopped by pressure. These tissues are so extremely vascular that they have a greater resistance against infection and necrosis than any other tissue in the body.

In a case I had some years ago I transplanted a large skin-flap from the neck, bringing it in from behind. We got only a fair opening of the mouth. The teeth had all been destroyed in that case. The procedure was much more difficult to carry out than this one, and the final result was not nearly so satisfactory

All the suturing in this case has been done with fine catgut. On account of the denudation here we have had to use two large-sized flaps. We have tried to put no tension on that, and, judging by the result obtained in a similar case operated on a few months ago, we are sanguine as to the outcome in this case. The one referred to was a little girl who had a fibrous ankylosis causing an absolute fixation of the jaw and inability to open the mouth. We interposed a mucous membrane flap, using only the upper or palatal flap. The result was a splendid one. When she left the hospital after five or six weeks she was able to open her mouth about one inch without any very great effort. We will pack this side of the mouth with nosophen gauze. That will not permit the jaws to come together. There are no raw surfaces to adhere and form cicatrices.

SUBCORACOID DISLOCATION OF THE HUMERUS WITH SEPARATION OF TUBEROSITY

HISTORY

Patient, male, aged thirty-three, comes to hospital on account of pain in left shoulder and inability to move arm at shoulder.

Present trouble dates back about two months, when the patient was thrown from a horse. The girth broke, and he fell, striking on his left arm, which was in the flexed position at the time. On rising he attempted to use his left arm, but experienced great pain in the shoulder, and extending down the arm. The pain in the shoulder increased in severity, and the patient was unable to raise the arm or to put his hand on the opposite shoulder. He could rotate the arm. The shoulder was manipulated under anesthesia and bandaged with arm against the chest. Pain in the shoulder continued, and extended to the elbow and to the extensor surfaces of the little and ring fingers. A second attempt at reduction was made and failed. The pain and the inability to use shoulder still continued.

COMMENTS AND OPERATION

Dr. Murphy: How many efforts at reduction have been made?

Intern: Two before he came here.

Dr. Murphy: We tried it a week ago, but failed. We were afraid of fracturing the arm. The artery was pulsating when we completed our efforts at reduction. We always make a note of that. One should always do that after attempting a forceful reduction of the shoulder. The artery should pulsate. If one has a record of the pulsation of the arteries and the arm becomes gangrenous later, it will be due to a traumatic endarteritis over which we have no control, and therefore the courts could not hold the surgeon responsible.

We believe that hematomata around the joint form in a large percentage of these cases later. That does not matter, however. It is the sudden ischemia from sudden arterial occlusion that causes the necrosis. That is beautifully illustrated in a number of specimens in the British Museum, which show the thoracic artery completely occluded in cases of Pott's disease by the abscess compressing the artery. These patients were never aware that the thoracic aorta had been completely occluded. The compensatory or anastomotic circulation rose to the occasion magnificently. That condition was illustrated very nicely in our experimental work on dogs. If we could keep up the circulation for four or five days, so that the occlusion occurred gradually, the tissues were not destroyed. That was Neff's plan. Neff worked out a method to produce a gradual occlusion of the vessels. He uses two flat pieces of metal bound together by a rubber band. On one end is a roll of chromicized catgut. As this is slowly absorbed, the rubber gradually compresses the plates and thus occludes the artery. In the mean time the collateral circulation has been developed and gangrene does not occur. This is based on the observation that a gradual occlusion permits a collateral circulation to form.

Dislocation of the shoulder occurs more frequently than of all the other joints in the body together. This is usually explained by the exposed position of the shoulder, its unusually wide range of movement, the range of leverage afforded by the humerus, and the anatomic construction of the joint, the comparatively large head of the humerus fitting imperfectly in the small and shallow glenoid cavity, and the ligaments being rather lax and thin. The capsule of the joint is strengthened above and behind by the tendons of the supraspinatus and infraspinatus and the teres minor muscles. The weakest part is below and in front, between the tendons of the subscapularis and teres major muscles. Therefore dislocations of the head of the bone occur most frequently in this position downward and forward.

The force of the blow is usually transmitted from the hand or the elbow with the arm abducted and the muscles relaxed. The head of the humerus is thus forced against the weakest part of the capsule; the capsule is ruptured, and the bone passes out through the rent into the subglenoid space.

There are several varieties of dislocations, according to the final resting position of the head of the humerus. The simplest is the subglenoid variety, in which the head of the humerus rests on the long head of the triceps muscle, just below the glenoid cavity. However, because the sharp head of the scapula and the round end of the humerus are not an anatomic fit by any means, this position is not long maintained. The bone continues in its course, depending on the nature and direction of the transmitting force and the position of the arm at the time of the injury, as well as the action of the muscles. As a rule, the bone passes forward and inward, so that it comes to rest on the anterior surface of the neck of the scapula, under the tendons of the biceps and coracobrachialis muscles. This is a subcoracoid dislocation.

Sometimes the bone continues in its course until it comes to lie under the pectoralis minor muscle against the edge of the clavicle—the so-called subclavicular dislocation. Sometimes, although not frequently, the head may pass backward instead of forward, and come to rest against the spine on the posterior surface of the scapula, under the infraspinatus muscle. This is known as the subacromial or subspinous type of dislocation. These dislocations or displacements may be attended with a fracture, and this may be a very serious complication, especially when it is the neck of the bone that is fractured, because then reduction is impossible without an open operation.

The subcoracoid dislocation usually results from a hyperabduction of the arm, with the scapula fixed, as in a fall on the inner side of the elbow. The surgical neck of the humerus bears on the under surface of the acromion, which forms the fulcrum, and the head of the bone is pressed against the inner and lower part of the capsule. Sometimes muscular action alone will produce this dislocation. A force applied directly to the upper end of the humerus will act similarly. The head of the bone passes through the rent made in the capsule in its lower part, and thus passes inward under cover of the biceps and the coracobrachialis till it comes to rest against the injured surface of the neck of the scapula, just below the coracoid process.

The guiding principle in reductions of all dislocations is to make the head of the bone retrace the course it took in leaving its socket. The obstacles to reduction are muscular contraction, the entanglement of the head with tendons, ligaments, or bony points. A general anesthetic is an invaluable aid to reduction, although the reduction may often be effected, especially in children, by simply gaining the confidence of the patient and keeping his mind off the dislocation, so that the muscles are relaxed. The limb may be manipulated without any additional muscular force being exercised by the patient. However, it must be remembered that too much manipulation should not be attempted in any case because of the frequency with which serious damage may be done to vessels, nerves, ligaments, and even the ends of the bones. In certain muscular subjects and in nervous patients, or when the dislocation is not a very recent one, an anesthetic should always be given. In quite recent cases, where the muscles are relaxed because of the shock or temporary faintness of the patient, reduction may be attempted without anesthesia and often is successful.

The plans of reduction used in dislocations of the shoulder are so well known that we will not attempt to discuss them at this time, but will give our attention more to the case at hand. There is no fracture of the head or neck of the bone in this case. The subcoracoid ligament has torn off a small fragment of the bone,—the tuberosity,—as is shown very distinctly in the skiagram. The dislocation in this case is of the subcoracoid type, but because of its comparatively long standing—two months—all efforts at reduction, even under complete anesthesia, have failed; therefore we shall resort to the open operation, exposing the joint through an anterior incision extending from the coracoid process downward and outward on the anterior aspect of the arm for a distance of 5 or 6 inches. Since our last attempt at reduction

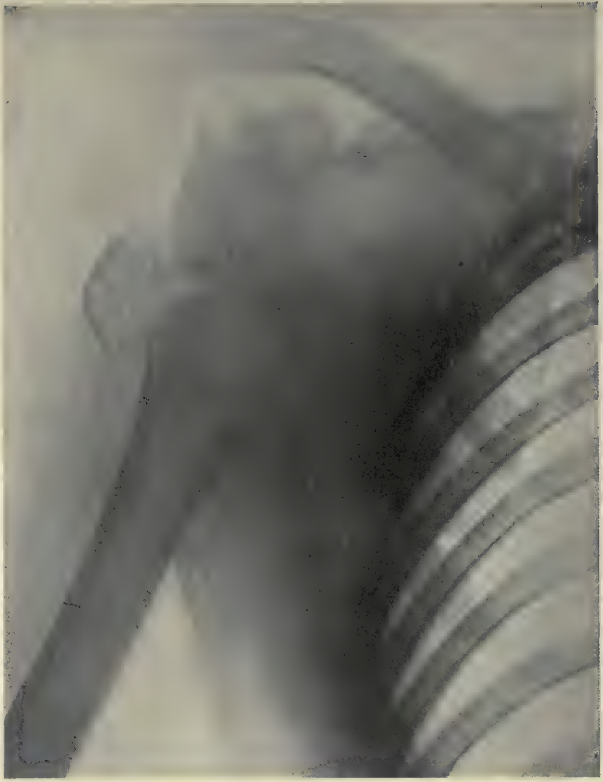


Fig. 80.—Dislocation of shoulder. Subcoracoid dislocation of the shoulder with a small fragment of the shaft torn off by the forcible contraction of the muscle. The fragment is discernible to the left of the humerus.

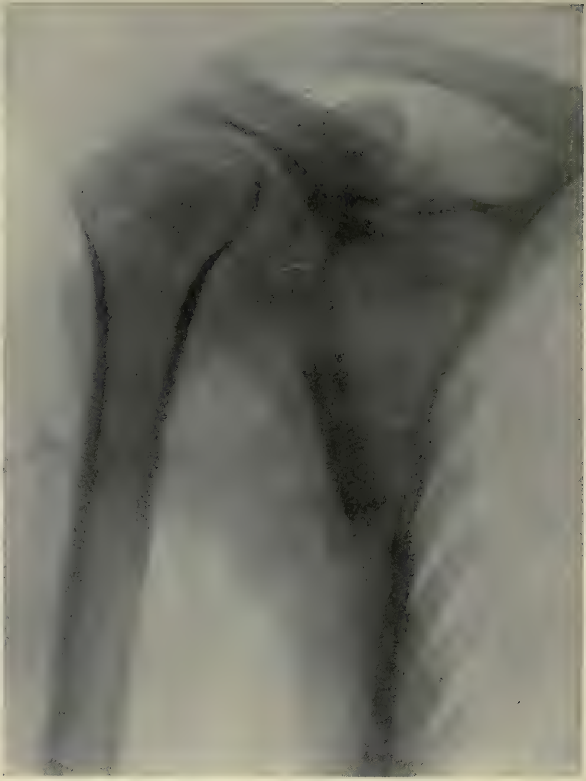


Fig. 81.—Shoulder replaced in position. Compare with Fig. 80.

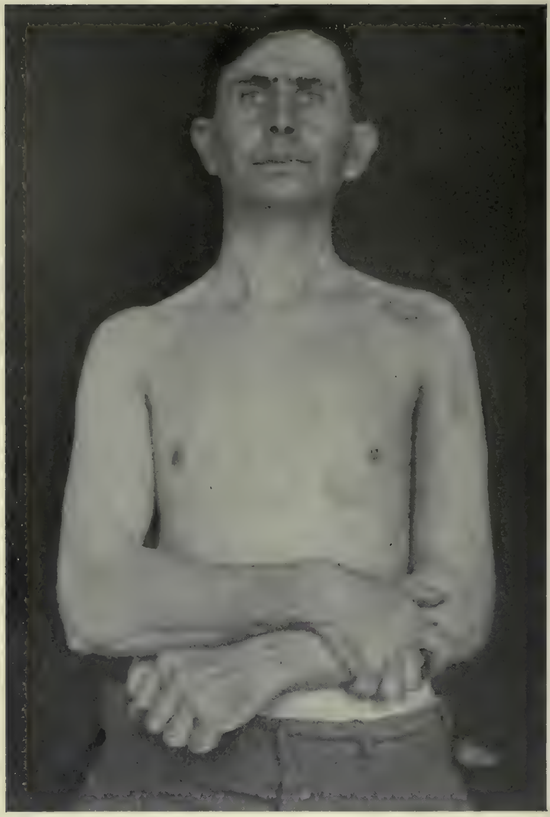


Fig. 82.—Normal contour of shoulder restored. The incision through which the operation was done shows very distinctly.

we have continued the extension on this arm so as to prevent a contraction of the muscles and to fix them in this position. As far as the muscular structures are concerned, we would like to expose this joint from behind. The operation certainly would be a much simpler one, and there is then practically no danger of injuring the vessels or nerves.

Looking at this arm, it would seem that the head of the bone is separated from the shaft, but that belief is not confirmed by palpation nor by the skiagram, and the head moves with the shaft of the bone. The dislocation is not a very bad one, because the head of the bone is not more than an inch away from its normal resting-place, but because of the length of time which has elapsed since the occurrence of this injury it has become fixed in its new position, and, therefore, it must be separated by an open operation.

I am making the incision between the pectoralis major and the deltoid muscles. I can feel the fragment of bone which was torn off from the humerus when the coracobrachialis ligament gave way. I can feel the shaft, the acromion process, the coracoid process, and the head of the bone. A fairly firm organic union has formed between the fibrous tissues, but the head of the bone is free.

We will first expose the glenoid cavity, which we find to be perfectly smooth and normal in outline. Next we will expose the head, and I find that in order to effect reduction in this case without injuring the surrounding structures I shall have to take off the coracoid process to which is attached the long head of the biceps and the coracobrachialis. I am dividing the coracoid process with the Gigli saw, leaving the muscles and ligaments attached to the fragment removed. It will be replaced before the completion of the operation. After this is accomplished the upper end of the humerus is clearly in view and easily controlled. So far as I know, this is a means not resorted to previously. I did not believe that I could slide the head back into the glenoid cavity without dividing the coracoid process and the long head of the biceps. The anatomic structures in and around this joint are well preserved, and we have no difficulty in recognizing them as they come into view.

The next step is the reposition of the head of the humerus in the glenoid cavity. By making extension with the arm in superlative adduction the head is pulled downward. Next, by rotating the arm outward and slowly abducting it until it is in extreme abduction, then rotating it inward again, we find that, with the assistance of two bone elevators used as levers, reposition is effected, although several attempts had to be made before this could be done.

You see it was rather difficult to restore the normal anatomic relationships in this case, even with the open operation, and no matter how long or how earnestly we had persevered in attempting reduction by manipulation, we never could have succeeded. This really was a splendid case for the open method, and I am glad to say that our efforts were entirely successful.

The coracoid process will be nailed in place with a six-penny nail. A small gutta-percha drain will be put into the bottom of this wound, to remain for seventy-two hours. The head rests easily in the glenoid cavity, and the arm movements are unrestricted. At no time during the operation were the vessels or nerves in the vicinity in any way endangered. The patient's pulse in the left wrist is beating normally. The skin incision will be closed with horsehair. The wound will be dressed with moist 5 per cent. phenol gauze. The arm will be dressed with a bandage only. It will be placed against the body with the forearm flexed at an acute angle to the upper arm, the hand placed flat on the chest, and fingers pointing toward the right shoulder.

[The patient had no pain after the operation at any time. The stitches were removed from the incision on the seventeenth day, and the drain was taken out in three days. The wound healed promptly. The patient was encouraged to perform passive motion in the shoulder. When he left the hospital, six weeks after the operation, he had good use of his arm, and, as he said, it was getting better every day.—Ed.]

FRACTURE OF NECK OF FEMUR: DISPLACEMENT OF HEAD ON DORSUM OF ILIUM

HISTORY

PATIENT, male, married, age fifty-one, enters hospital on account of limited motion, weakness, and shortening of left leg. November 18, 1912,—three months ago,—the automobile in which he was riding ran off the road, turned turtle, and rolled down a 70-foot bank. He was thrown out, but does not know how he landed or how he was hurt. He could not move his left leg. It was partly flexed, the long axis of the thigh being directed inward toward the opposite leg. He was taken to a hospital at once. After twenty-four hours he was given an anesthetic and an attempt made to reduce his hip, which they told him was dislocated. An extension of 8 pounds was put on and kept on for seventy days. He had considerable pain at first, but it gradually subsided. After the effort at reduction, the leg could rest flat on the bed in the extended position. One week after the extension was taken off he began to walk on crutches. He has not put any weight on the leg to the present time. Motion in all directions is limited. He seems to have no power to lift the leg, although he can rotate it fairly well. Has been gradually improving. Had no motion in knee at first, but now he has fairly good motion in it. Has very little pain in either hip or knee. Claims legs were same length when extension was put on, but left leg is one inch shorter than the right now. History is negative otherwise.

General health always good—never any sickness. Examination of the skiagram shows a fracture of the neck of the femur, with the dislocated head resting on the dorsum of the ilium, just behind the acetabulum. It has a spicule of bone projecting from it. There is a shadow in the acetabulum, as though cast by a fragment of bone—probably a piece broken off from the head or a deposit of bone. The shaft is displaced upward about an inch or an inch and a half.

COMMENTS AND OPERATION

Dr. Murphy: Nearly three and one-half months have elapsed since the injury. He had an extension on for seventy days, or until just recently, which prevented the shortening. It is very important to keep up the extension if you are going to make any attempt at reduction. When these muscles that are attached to the femur are allowed to contract and become fixed, it is difficult to make a secondary elongation. That is a very serious obstacle in making the bloody or unbloody reduction of congenitally dislocated hips in children—that after the age of six or eight the muscle contraction is very great. That is one of the factors that we will have to overcome in this case. If the head is entirely separated from the shaft and free, we can easily put it back in the acetabulum. That will be an easy proposition. The head may have united with the neck, notwithstanding that the skiagram shows it to be free, because in the early months of bone regeneration the *x*-rays are not obstructed by the newly formed bone. They go through, and it looks as though the fragments were not united, when in reality they are united. It would be hard to make the reduction with the head united to the shaft unless we refractured the neck. In the case of the shoulder we have no trouble with that factor. We separate the misplaced head from the shaft, take it out on the table, shape it, and put it back in its proper place. We believe that in the hip regeneration should take place just the same as it does in the shoulder, but we have not had sufficient clinical results to demonstrate this to a certainty. We have had only one case of that kind so far. (See February, 1913, Clinics.) One case establishes a principle, but you must have more than one successful case positively to establish a practical fact. We also had a successful transplantation of the trochanter major on to the neck of the femur following fracture where the head had been absorbed. (See February Clinics.)

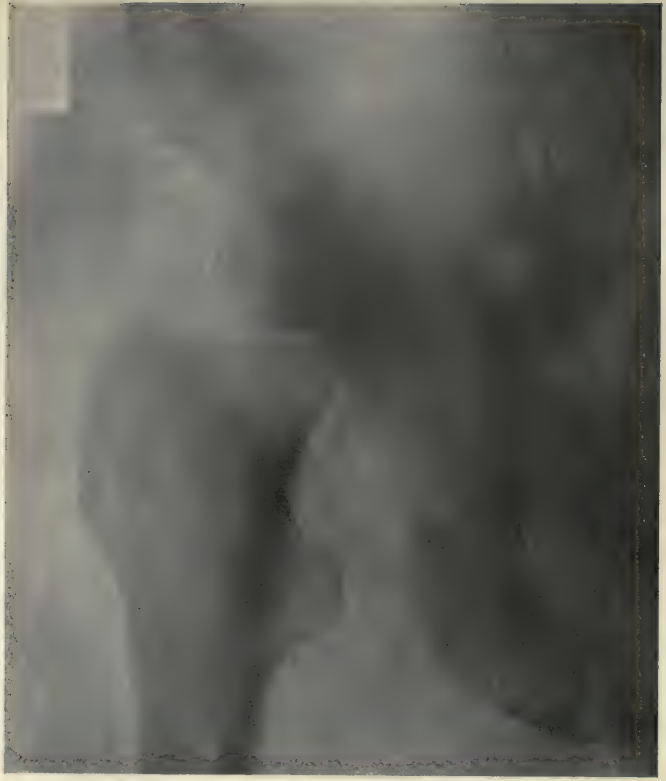


Fig. 83.—Fracture of the anatomic neck of the femur close to the head. No absorption of bone, but head of bone is rotated and dislocated upward out of the acetabulum, resting on the dorsum of the ilium, just above the upper rim of the acetabulum.

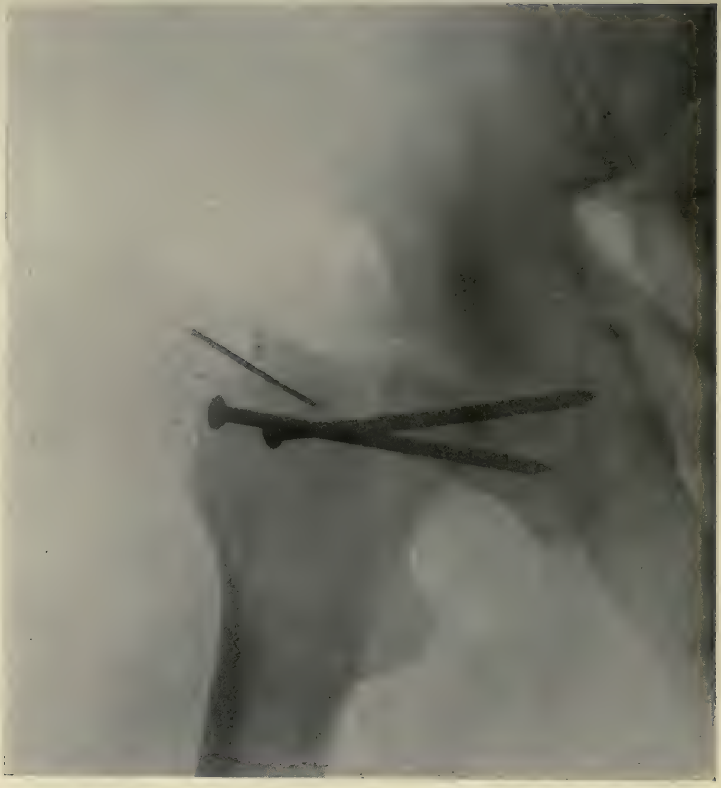


Fig. 84.—Two weeks after operation. Fragments in good apposition and the head of the bone in the acetabulum. Two large nails secure the approximation of the fragments. The small nail passes through the trochanter. Phosphor-bronze wire sutures are visible in the soft parts around the trochanter.

We are going to proceed on the same plan in this case as we would if we were going to do an arthroplasty. Looking over the anatomy of the hip, the one fact that impresses itself on you is that there are no muscular attachments to speak of on the anterior surface of the neck. They are all on the trochanter of the femur. The muscles all come from behind and above. Coming from in front are the muscles of the adductor group, the adductor longus, magnus, and brevis, and the gracilis. These muscles adduct the thigh. They are very powerful, and often contracted in these cases. If you flex the thigh, you can take off some of the tension on these muscles. The next group of muscles to be considered is the psoas and iliacus muscles. They are attached to the lesser trochanter from behind, and they react to extension and rotation. They flex the thigh on the pelvis. The one muscle that has no relaxation is the gluteus maximus. It extends the thigh. The gluteus maximus we do not need to detach. The obturator externus and internus, the gluteus medius, pyriformis, and gemelli muscles are attached to the tip and side of the great trochanter. We are going to take off the great trochanter, with its muscular attachments, so as to be able to locate what is left of the neck of the bone and to inspect the acetabulum. By dividing the trochanter with a chain-saw and retracting it upward the hip-joint is freely exposed and accessible. The acetabulum in this case should be perfectly free, because it is only three months since the injury, although the skiagram leads us to believe that the acetabulum is not free. If the head is entirely separated from its normal attachments, it takes on all the characteristics of a bone transplant, and osteogenesis will proceed from the neck into the head, and a new head will be formed, as occurred in one of our cases. If it is a fracture at or near the epiphyseal line absorption of the bone will take place, and go on centrifugally for a long time unless union is established. If the fracture is near the trochanter, away from the epiphyseal line, absorption will not take place centripetally. Of course, friction also plays a rôle in the absorption.

I remember a patient from New York who had a compound fracture of the neck of the femur which four doctors saw. Three said it was not fractured and one said it was. It was an impacted fracture of the femoral neck. The man was permitted to go about, and at the end of three months the leg was materially shortened; at the end of two years and four months—the time I saw him—he had no femoral neck at all, and only a button of a head in the acetabulum. This was an interesting case, and shows how the absorption takes place from within outward. (See February, 1913, CLINICS.) The vessels go in from the neck to the base of the head, and turn around and come back in the bone. When the neck breaks just at a line one-half inch outside of the epiphyseal line, absorption takes place. It is the result of defective circulation. Whether that is the correct solution remains to be ascertained. We believe it is correct.

We had another case of fracture of the femoral neck at the base of the trochanter a year and three months before the operation, and there was no absorption at all. That man was here thirteen or fourteen weeks, and made a good recovery. These two cases are very valuable and forceful clinical lessons. Both of them occurred about the same time, and both came into the hospital about the same time after the accident. They are contrast cases.

This patient is fifty-one years of age. He has always been a very active man and a man of good habits. If the head is completely detached, I may have to take it out and put the neck of the bone into the acetabulum, using it as the support for his leg. Generally, motion is limited in such a case at the beginning, but there is gradually developed more and more motion and the final result is fairly satisfactory. The other alternative is freshening the bone surfaces, replacing the head in the acetabulum, and nailing it to the neck.

We have had very nice results in our osteoplastic work so far. We are going a little farther in each case, as we feel we should. We have four cases of fractured femoral necks in the hospital at the present time. We do the operation in a very simple way. We make a skin incision, beginning above and in front of the trochanter, parallel to the fibers of the gluteus medius anteriorly, and incircle the trochanter, making a U-shaped skin, fat, and fascia flap. The muscles are not cut, except sometimes a few of the anterior fibers of the gluteus medius. Expose the neck fragment, get it in the proper relationship with the head, and fix it with one or two ordinary wire nails. This does not require much manipulation. We cannot do that in this case, however, because the head of the bone is luxated out of the acetabulum.

The period of time these patients are kept in bed after the operation is usually from fourteen to sixteen weeks. They are put in a travois splint, with both legs abducted and in the superlative degree of extension. Early cases of fracture of the neck of the femur are treated by abduction and extension. Remember, that you cannot abduct one limb only, because when you abduct only one limb, the patient moves his body over to the other side, throwing his body in a straight line with the broken hip, thus abducting the well leg. That is why the travois splint fits so nicely in these cases. Both legs are abducted and remain so.

The head of the femur in this case appears to be behind the neck. I am now making the U-shaped incision, as described above, and retracting this skin and fascia flap upward; the trochanter is in view. I am endeavoring to pass the chain saw under the muscles attached to the trochanter. The trochanter will now be sawed off and, with its attached muscles undisturbed, it is retracted upward. There is the head of the femur. It is entirely detached from the femur, out of the acetabulum, and resting on the dorsum of the ilium. I am detaching the Y or iliofemoral ligament, cutting down to the neck. Next I will split the capsule so I can expose the neck. It was attached to the highest roof of the neck. The top of the neck is in plain view, but the acetabulum is not in view at all. It appears as though there might be a part of the tip of the head left in that acetabulum. I am looking for the fractured surface of the neck. It ought to be in the acetabulum. I do not want to do anything here until I get this acetabulum cleaned out. The tissue in the acetabulum feels like cartilage and looks like cartilage. It is in all probability a portion of the articular surface of the head.

Now I am going to see if I can put that neck into the acetabulum so as to give support to the leg in case we cannot make use of the head. There is the head, on the dorsum of the ilium, with connective tissue between it and the neck. The tissues are partially united to the side of the fractured acetabulum. The head ought to come right out, but it does not, because of this new-formed fibrous tissue. With careful scissors dissection we free the head from its bed, grasp it with heavy bone forceps, and remove it entirely from the body. A portion of its articular surface is missing. That is the piece of bone and cartilage in the acetabulum. The capsule which is attached to the margin of the acetabulum will be turned in to fill up that acetabulum. That will effectively prevent an ankylosis from occurring later.

The piece of bone in the acetabulum will have to come out before the head can be replaced. That grew in much firmer than I believed it would in three and one-half months. That should not have grown in so solidly in that period, but it has.

Now the acetabulum is cleaned out, and the capsule fragments are turned into it. The fractured surfaces of the head and neck will be squared off with bone-cutting forceps, so that we will have two good apposing surfaces. By adducting the leg and turning or rotating the knee outward the neck is easily thrown out into the field of operation. Now we will replace the head in the acetabulum. This is only the second time we have done this. By extending the leg slowly, abducting it, and rotating the knee inward, we secure a good approximation of the head and neck. Two wire nails are then driven through the neck into the head from the shaft side, and the operation is completed. I did not overestimate the difficulty in this case. The trochanter is replaced, and a small wire nail is driven through it into the shaft.

The reconstruction is not nearly so severe as one would expect it to be. The deeper layers of the wound are closed with phosphor-bronze wire. Horsehair is used to close the skin incision.

Let the record show that, after exposing the trochanter, the head was found resting on the dorsum of the ilium, posterior to the acetabulum; that it was permitted to remain until we had cleared out the acetabulum. On examination of the acetabulum it was found that a portion of the head had been fractured off and remained in the acetabulum. This was excavated. The head was then freed from its position, taken out on the table, including a portion of a fragment of the neck attached to it, was squared off so as to get a broad surface to rest against the freshened neck. A point of the neck was also leveled down to adjust itself to the head. It was a little difficult to replace the head in the acetabulum, but finally it went in in good position, and there was an accurate approximation of the head to the neck, as is shown in the skiagram (Fig. 84). The fragments were held in apposition by two twelve-penny wire nails driven in from the shaft. The trochanter was replaced and secured in position with a small wire nail. We then sutured the fibrous structures with phosphor-bronze sutures. There were no ligatures used in the entire operation; the oozing had practically ceased before we were ready to close the wound. The whole operation involved a considerable dissection. Both legs will be placed in a travois splint in supreme abduction without extension.

VISITING DOCTOR: How long before you will dress that wound?

DR. MURPHY: If all goes well, in about three weeks. The purpose of the moist 5 per cent. phenol gauze which we always use in this class of cases is to take up the secretions from the wound and keep them sterile. It prevents the growth of the saprophytes. When you remove this gauze in three weeks there is absolutely no odor. I remember seeing Lister taking off this old-fashioned 5 per cent. phenol gauze and asking his audience to smell it. It showed that he was right in his theory. The danger of infection is not so great in this as in the earlier cases. If all goes well, he will be back to normal in three months.

[Dr. Edward Martin and Dr. Robert Le Conte, of Philadelphia, were present at this operation. The wound healed per primam. The man has been free from pain and has not had an untoward symptom. He will be kept in bed for fourteen weeks. Then a skiagram will be taken to determine the degree of osteal reproduction.—ED.]

FRACTURE AND DISLOCATION OF SCAPHOID AND SEMILUNAR BONES

HISTORY

Patient, male, aged twenty-nine, enters hospital on account of an injury to left wrist, the result of an accident which occurred eighteen days ago. While holding on to the top grab-iron of a box-car, it was struck by an engine with great force, causing him to relax his hold with his right hand, and throwing all his weight suddenly on his left wrist. His feet were knocked off the baseboard by the contact. His body twisted around on his left wrist in a position of abduction. Then he released his hold and fell to the ground. He had no use of his wrist at all immediately following the accident nor since. Soon after the accident he noticed a mass above the wrist on the anterior surface. A temporary splint was put on. The next day he was given an anesthetic, and an attempt at reduction was made. It failed. Then a well-fitting tight splint and a roller were put on, causing him great pain. The bandage was so tight that his fingers were numb. This numbness gradually wore off. The dressings were kept on and he was brought to Chicago February 21, 1913.

Examination.—The skiagram indicates that the scaphoid bone of the left carpus is fractured and half of it is luxated. It is displaced upward anteriorly, one-half inch, resting on the lower end of the radius. The semilunar is completely dislocated. There is a concavity below the lower end of the ulna and radius, with only the pisiform contacting with the ulna, and a part of the scaphoid is contacting with the radius. The bones of the second or upper row of the carpus are disturbed in their normal relationship. By the removal of these bones through an anterior longitudinal incision and separating the tendons, and without opening the tendon-sheath, his wrist can be restored to its former usefulness. The bones cannot be replaced. There is a concavity on the posterior surface of the carpus, due to the luxation of these bones.

COMMENTS AND OPERATION

Dr. Murphy: This history is a rather interesting one, because it shows so well just how the injury occurred. His left hand supported the entire weight of his body when his right grip was torn loose, and then the turn made by his body threw his hand back and his wrist forward, so that the lower end of the radius and ulna did the damage to the carpus.

This is the first case of this kind that I have seen, although dislocations of the carpal bones are hardly surgical curiosities. I have seen one case where one of the carpal bones was completely luxated. In another case two bones were partially luxated. In the case of the patient before us the semilunar bone is completely luxated, not only from the carpus, but up onto the radius; the scaphoid is fractured, the upper half being dislocated. These two bones are underneath the flexor tendons of the forearm. The fracture of the scaphoid is very evident. This type of accident often produces a luxation of the os magnum, either anterior or posterior, but in this case one can readily draw a mental picture of just how the injury resulted. Codman, of Boston, some years ago wrote a very fine paper on fractures of the os magnum and scaphoid, showing how they were responsible for useless wrists.

This patient's hand looks normal, and when his wrist is covered, there is nothing to lead one to suppose that anything is wrong. When you look at his wrist, there is a small tumor at the lower end of the anterior surface of the radius, and on palpation it may be concluded at once that it is a bone. By referring to the skiagram the diagnosis is readily made, but it could not be made without the skiagram.

There is a decided depression below this tumor. It is caused by the absence of these bones from the carpus. I can easily put my finger into this cavity. I will make a longitudinal incision on the anterior aspect of the wrist, just to the inner side of this tumor, which is pressing against the tendons, immobilizing them, and rendering the hand useless. We must be very careful in our dissection not to cut into the tendon-sheath nor to injure any vessels or nerves. The median nerve is just outside of the tumor, and as we continue with our dissection it is easily recognized and differentiated from the tendons, because the tendons have a pearly-white color and are glistening, whereas the nerves have a pinkish color and do not glisten.

I displace the median nerve to the inner side, and then continue my dissection through these groups of tendons until I reach the bones. The articular surface of the semilunar bone is directed upward. This bone has become rotated. I can remove it very easily. Using the excitor, one finds that the nerve quickly responds. The fragment of the scaphoid also comes out easily.

We have had very little hemorrhage here, and we are now ready to close the wound. The tendons, you will note, were not injured in any way. A subcuticular catgut stitch and horsehair skin sutures closed the wound. We did not put in a drain. The arm will not be splinted nor placed in a cast.

[The subsequent course of the case was uneventful. The stitches were removed on the tenth day. When the patient left the hospital, three weeks after the operation, he had good use of his wrist.—Ed.]

DISLOCATED SEMILUNAR CARTILAGE DISPLACED ACROSS MEDIAN LINE OF JOINT

HISTORY

Patient, male, aged twenty-nine, enters hospital on account of recurrent attacks of pain in and incapacity of left knee. Trouble dates back to September, 1911, seventeen months ago, when he slipped and fell, striking his knee on a steel rail. During that day he had several twinges of pain in the knee, but the knee did not interfere with his work. The next day he noticed that on taking a sudden step he would have an acute pain in his knee, and it seemed to lock. Then, by taking his weight off that leg, and with a little manipulation of the knee, it would be all right again. This condition gradually got worse, until he was unable to take a single step without the knee locking. His knee began to enlarge after each locking and swell slightly and be very painful, until, after two weeks, the joint was very large and painful. He entered the hospital, where injections (?) were made into the joint. Condition was better for several months, when he slipped again and fell, dislocating his hip and fracturing the neck of the femur. After the rest in bed demanded by the treatment of the fracture his knee seemed better for some time afterward, but now he feels the knee lock again when he flexes it suddenly, and it swells and becomes painful for several days. He now uses a crutch all the time to keep his weight off the leg. Contracted lues in 1911.

COMMENTS AND OPERATION

Dr. Murphy: Looking at the skiagram, there appears to be something in the track of the x-ray. It does not look like a normal semilunar cartilage. Looking at it the second time, it seems as if he might have had a fracture of the internal tuberosity. The bone level on the inside is a little below the bone level on the outside. He first had the locking of the knee; following that he had edema and swelling, and following that he had an effusion into the joint. The external condyle of the femur is, if anything, a little high. The internal condyle is not on a level with the external condyle. That is what makes me think that when this man struck the rail the first time he had an impacted fracture that took off a corner of his tuberosity. That is about the way it looks when he flexes the knee suddenly.

He had lues in 1911. That must be taken into consideration. A spirochetic infection is a dangerous factor in all traumas of joints. As you look at that skiagram you will notice that the internal condyle, the internal tuberosity, the internal half of the joint, is lower than the external. That means that he had a pathologic process going on in the joint, in the cartilage, in both tuberosities and condyles, the result of recurrent traumas. The most important feature that I wish to bring out is that the joint is not movable. If he had a depression of his tuberosity, as it appears he has, he should have an impingement of the internal tuberosity against the femur, and that is why he would have an immovable joint.

Luxations of the semilunar cartilage occur much more frequently in the internal cartilage than in the external; that is, in a proportion of 50 to 1. If we simply take the story of the trauma, we would consider this an ordinary case of luxation of the semilunar cartilage, but he has an additional complication here. The condition shown in the picture might be from his trauma or from his spirochetic infection. There is some fixation of that joint. The internal condyle is a little higher than normal.

I will make my incision close to the ligamentum patellæ, on the inner side, going below the tuberosity so as to give me a little better view of this joint. I am cutting upward right along the margin of the quadriceps tendon. The internal tuberosity stands out much more prominently than normal. Either that is due to the spirochetic infection or it is the result of the repeated traumas. We may be able to determine that later. Note what a great quantity of tissue there is between the capsule and the

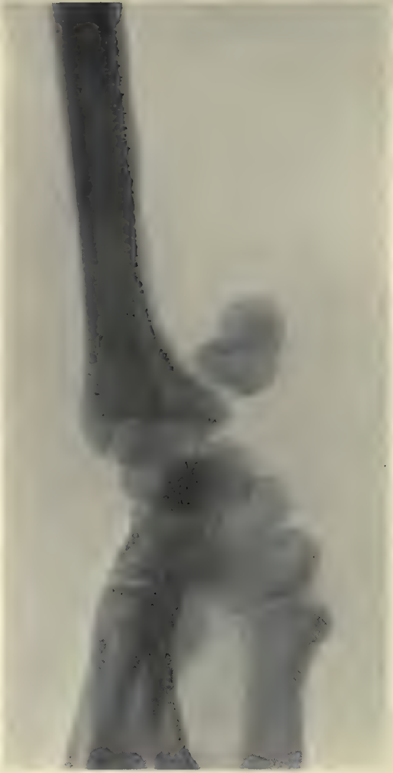


Fig. 85.—Luxation and fracture of carpal bones. Lateral view, showing the change in position of all the carpal bones, with a dislocation of apparently only one bone on the anterior surface of the lower end of the radius.

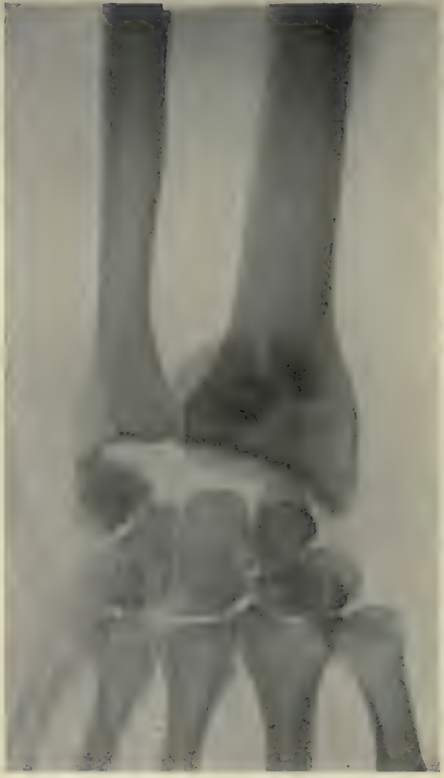


Fig. 86.—Anteroposterior view. Dislocation of the pisiform bone on the anterior surface of the unciform. Dislocation of the semilunar upward on the radius. Fracture of the scaphoid, with a dislocation of the upper fragment on to the anterior surface of the radius. The normal anatomic arrangement of all the bones of the carpus, except the os magnum and the unciform, is destroyed.

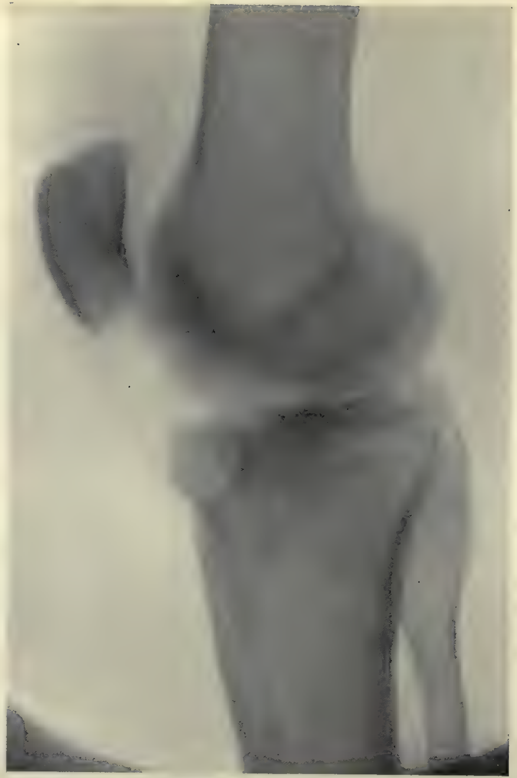


Fig. 87.—Dislocation of the semilunar cartilage. Lateral view of the affected joint, showing the irregularity in the upper surface of the tibia from which the semilunar cartilage was dislocated and fractured.

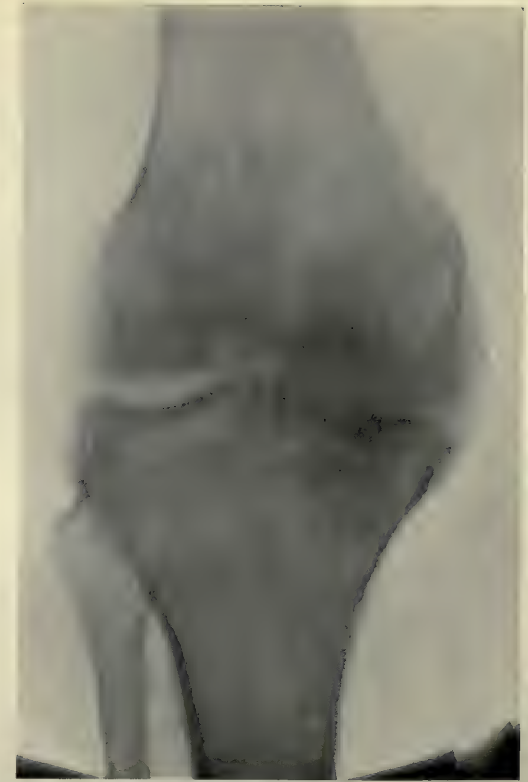


Fig. 88.—Anteroposterior view, showing the internal condyle of the femur in close apposition with the internal tuberosity, from which the internal semilunar cartilage is dislocated. Obliteration of joint cavity on inner side.

synovial membrane. Now we are coming into the joint, and there is the synovial fluid. I do not wish to injure the synovial membrane, because it might lead to an ankylosis. That is not a normal cartilage. It corresponds exactly with the skiagram. The cartilage is in my field in plain view. Some ossification has taken place on the surface of the inner tuberosity. The whole field is more vascular than normal. There is a strip of the cartilage worn by repeated friction down to a point. That cartilage has fractured or split and half of it thrown over into the middle of the joint. I have loosened it. It shows that the pathology is in perfect consonance with the history. There is a cicatrization and thickening of the semilunar cartilage, so that it is very much harder than the average semilunar cartilage, due to the constant irritation of the trauma. It is entirely traumatic, and not the result of the spirochetic infection. Instead of being a soft, smooth cartilaginous tissue, it is hard and thick. You can all see the change in the cartilage. See what a different picture that is from the normal cartilage! The normal cartilage is all destroyed by the repeated traumas.

The synovial membrane is also thickened. The reason I do not fear infection in this case is that the synovial membrane is ready for resistance against infection, as if I had injected formalin and glycerin.

You will note that in handling that knee I allow nothing to enter the joint but that sponge and the instruments. That is the key to keeping the field sterile. The outer layers of the synovial membrane under the fibrous capsule become infected first. The inner layers become involved only after the other tissues have all been destroyed.

Having removed this cartilage, I am ready to close the wound. The fibrous capsule will be sutured with plain catgut, and the skin with horsehair. The capsule is very thick. You can see why, when infection takes place in that joint, it is held under great pressure because that fibrous capsule has very few lymphatics and no vascularity, and it is firm, like a leather bottle. That is why you do not get edema and swelling of the tissues outside. That is why you do not have an early escape of the infective microörganisms from within the joint out into the surrounding cellular tissue. That is what occurs in infections in other portions of the body. This is an inelastic white fibrous capsule that stands a hydraulic pressure of from 20 to 100 pounds and more before it ruptures, and it is not rapidly destroyed because of its non-vascularity. That is why, when we inject the joint with infective micro-organisms experimentally the joint does not become infected unless we have destroyed the endothelial layer of the synovial membrane so as to admit microörganisms into the subendothelial lymph-spaces and vascular channels. We know that the synovial membrane, the subsynovial membrane, the cellular tissues, are very easily infected and rapidly destroyed on account of their vascularity. You can see what an enormous destruction has taken place within before the outer wall gives way. That is how joints are destroyed, differing from the infection in the cellular tissue in other portions of the body.

A Buck's extension will be put on this leg and kept on for three weeks until all the reaction to the trauma has subsided. He can carry about 20 pounds. This zinc oxid plaster irritates the skin. The old diachylon plaster was the ideal plaster for the skin. All the zinc oxid plasters irritate the skin. They are especially irritating if you have to sterilize the skin with iodin. If you use the old diachylon plaster, there is no irritation of the skin. I am endeavoring to get a plaster to take the place of this one. [Such a plaster has since been found. It is made by Charles Shivers, of New York. It is known simply as an adhesive plaster.—Ed.]

Let the record show that the central portion of the cartilage covering the internal condyle was thin and worn out and almost destroyed. This was due to the fact that when the cartilage fractured and stretched, it rested across the middle line of the joint. That is why he had contusion after contusion in place of a stretching from side to side. That has also produced a similar change in the cartilaginous surface of the internal tuberosity. The condition resembled an acute inflammatory infection of the joint. There was no velvety condition of the synovial membrane, and no evidence that the patellar tubercle was contused and fractured. I never saw a cartilage going across the middle line before. It was fixed at the tip and loose at the base, never going in and out. There was a continuous pressure on the cartilage, and you can see that no amount of local treatment could ever give any relief. [Healing by primary union. Patient left hospital in four weeks.—Ed.]

INFECTIOUS GRANULOMA OF THE CAPUT COLI—RESECTION OF THE CECUM AND ANASTOMOSIS OF THE ILEUM TO THE ASCENDING COLON

HISTORY

PATIENT, male, age forty-four, enters hospital on account of colicky pains in the epigastrium, belching of gas, and a hernia in the right lumbar region. September 2, 1912, the patient had an acute attack of appendicitis, and on the next day was operated on. The appendix was gangrenous, and free pus was present in the abdominal cavity. This was drained, the tube being left in about three weeks. Four weeks after this operation, and while still in bed, he was operated on for hemorrhoids, which were causing him great pain. At the end of the eighth week he left the hospital, feeling weak but in fairly good condition. The latter part of November he noticed a hernia appearing at the site of the abdominal wound. This has remained about the same in degree up to the present time. He is always tender around this region, but otherwise has no symptoms.

January 23, 1913, about five weeks ago, had a severe colic in the epigastrium. It came on suddenly and lasted about two minutes, when he was able to belch up some gas. This relieved the colic. These attacks kept coming on every three to four minutes the remainder of the day, and have continued up to the present time. He has not vomited nor even been nauseated during these attacks. Has been on a liquid diet because if he eats solid food the colic becomes so severe and his stomach so full of gas that the distress and pain are unbearable. When he does not eat anything, the symptoms are not so severe but do not disappear entirely. He is constipated, but there never has been any blood in the stools to his knowledge.

COMMENTS AND OPERATION

Dr. Murphy: Where was the pus, inside or outside of the peritoneal cavity?

Intern: It was a peri-appendiceal abscess.

Dr. Murphy: How soon after the onset of the symptoms did the abscess rupture?

Intern: Six hours.

Dr. Murphy: That is a very rare occurrence. It is barely possible that this was a case of enterolith in the appendix which ulcerated through the wall of the appendix and was not found at the operation. That has happened more than once. He has had symptoms that simulated appendicitis since the operation. He had two attacks before he came here. He entered the hospital in the third attack. If I did not know that his appendix was out, I would say that he has appendicitis now. On palpation of the abdomen we found an infiltration of the caput coli. Whether that is an acute inflammatory infiltration or whether it is a tuberculosis, or an old peri-appendiceal abscess that has formed slowly from a fecal concretion lodging in a mass of adhesions we cannot say. That mass at first seems to be as large as my hand, but on slow manipulation it gradually diminishes in size until it is no larger than my finger. How high was the temperature in this last attack?

Intern: 102° F.

Dr. Murphy: What was the leukocyte count on admission?

Intern: Twenty thousand.

Dr. Murphy: That may mean that he has a malignant lesion of the caput coli. Again, it may be a tuberculosis originating in the appendix. He is a man of fairly good habits. The Wassermann reaction was negative. His leukocyte count has been dropping down almost constantly since he came here. His temperature has been rapidly diminishing but he is still suffering, notwithstanding that the tumor mass had diminished in size fully 80 per cent. We are going to expose his caput coli and do whatever is necessary.

These fecal concretions in the appendix often give quite a bit of trouble. The second case of appendicitis in my experience was one with a fecal concretion in the appendix. I had operated in the morning at Cook County Hospital, doing my first appendectomy that day—March 2, 1889. When I got through with that operation they asked me to come over to the medical side and see a case of typhoid perforation. The girl gave a history of a typhoid. After recovery she had been apparently well until shortly before I saw her. She was scrubbing the floor four days before. The floor was wet, and when she got up from her knees she slipped and fell, landing on her right side. Immediately she had sudden pain in the abdomen. The pain got steadily worse, and was very severe when she was brought to the hospital three days before I saw her. The story she gave me was that she began to vomit after the onset of the pain. I had just read Krafft's famous article on the surgery of periappendiceal abscess due to the appendix, and was familiar with Fitz's article on the medical phases of infections of the appendix—the two great papers on the appendiceal problem. Krafft wrote his article in 1888, two years after Fitz wrote his. I made a diagnosis of ruptured appendiceal abscess, basing the diagnosis on Krafft's analysis of his cases. Krafft had collected the histories of his cases and reported them as pathologic curiosities. I was positive that this girl had had appendicitis one year before and not a typhoid, as was said. We operated and drained a diffuse peritoneal abscess. She was then moribund. She died four hours after the operation. Rather high up in the appendix was a stone—a wedge-shaped stone. It rested half in and half outside of the appendix. Outside of this was a circumscribed, dense-walled periappendiceal abscess which evidently had been present for a year. On the under margin of this was a small hole, showing a recent rupture, which permitted the pus to escape into the free peritoneal cavity and this caused the general peritonitis which was present at the time of operation. Evidently, when her foot slipped, the contraction of the abdominal muscles caused the abscess to rupture. I never had an appendicitis case before that time, and I had two in one day. That was my initial experience. The fact that the stone stayed partially in the appendix and partially in the abscess this long period of time is one of the points to be remembered in connection with this case.

The pain in the case before us occurred in the caput coli region. It occurs much more commonly in the sigmoid and descending colon, but more commonly in the sigmoid. The temperature dropped rather suddenly on the second day of the attack. He had a high temperature for three days, then it dropped suddenly and it has been normal ever since.

When he is thoroughly relaxed, there is quite a mass here. I am making my incision to the inner side of the old incision, going through the right rectus muscle. There are extensive adhesions present. I do not know whether this is the sequence of an infection, the sequence of the drainage, or the sequence of a tuberculosis. Those cobweb adhesions are always suspicious. They occasionally occur, however, in an inflammatory condition, entirely independent of tuberculosis. Now we are coming along to the position where this mass is located. I am going to separate these adhesions carefully with my finger. There are large pockets of pus here. There is a fecal discharge. Sometimes you get a number of these abscesses emptying into one pocket. I remember seeing as many as 25 emptying into one pocket. There appears to be a neoplasm or ulcer down in here involving the anterior surface of the cecum. There is quite an extensive infiltration; in fact, it is a firm, woody mass, so that I believe we are justified in removing the caput coli entirely, making an approximation between the ileum and the ascending colon. We will free the bowel from these dense adhesions in which it is embedded, and exsect about four inches of the colon, including the caput coli. The bowel is clamped transversely, divided with the knife flush with the clamp, and the stump cauterized with the actual cautery. The stump will be embedded exactly the same as we embed the stump of the appendix. Two rows of sutures of Pagenstecher linen will close the bowel. The first or innermost row is of the Czerny-Lembert type, and includes all the coats of the bowel. It is an in-and-out stitch. Each stitch goes through all the coats. The second or outermost row is of the Lembert type, including only the serosa. It is a reënforcing suture. That completes the inversion. Next we will divide the ileum about two inches to the left of the ileocecal valve, in the same manner as we did the cecum, and now we remove the mass.

The mass is rather firm in consistence, and measures about 2 inches in length and 1½ inches in its greatest diameter. It may be a sarcoma or a tuberculosis. On the other hand, the infectious condition from which this man suffered previously may be the cause of all this trouble, so that we must think of an infectious granuloma before making a positive diagnosis of the mass. It gives with not much difficulty, and it looks like a cellular mass of tissue. That means we will have to differentiate between a sarcoma and an infectious granuloma. So far as we are concerned, however, we are entirely justified in removing the tumor, because the patient could not have been given any relief from the obstruction in any other way.

The caput coli was enormously infiltrated, and it was surrounded by many dense adhesions. It was impossible to differentiate between a carcinoma, granuloma, sarcoma, or tuberculosis; therefore no other course was open to us than to make a resection. We feel that this man will be entirely relieved from his symptoms of obstruction so that the end fully justifies the means.

We will anastomose the divided end of the ileum with the side of the ascending colon—a typical end-to-side suture anastomosis. This will be done in the usual manner, making the opening as large as possible, so that any contraction which may occur subsequently will not interfere with the free passage of the intestinal contents from the small into the large bowel. There is no hemorrhage here, because the cautery has closed the ends of all the vessels. We will not use the button, simply making a suture operation. Pagenstecher's linen is the material used. That makes a very satisfactory operation, not only for the operator, but for the patient. The hole in the mesentery will be closed with a puckering-string catgut suture. Because of the infection present in this case the abdomen cannot be closed without drainage; therefore a rubber tube drain about as large as a lead-pencil will be inserted through the wound down into the abdomen, in the position behind the line of intestinal approximation. We will also pass a rectal tube and keep it there for two or three days. The peritoneum will be closed around the tube with catgut, making an ectropion of the edges of the incision. Catgut figure-of-8 and silkworm-gut sutures complete the closure. The point of the drainage-tube will be fixed in position with a silkworm-gut suture.

[The patient made a rapid and uneventful recovery. He had no pain after the operation and no distress. The rectal tube was removed on the fourth day, and the peritoneal drain was removed on the eighth day. The abdominal wound closed primarily, and the patient left the hospital entirely well three weeks after the operation. Microscopic examination of the tumor mass showed it to be a typical infectious granuloma.—Ed.]

ARTHROPLASTY OF THE HIP—TROCHANTER PLACED IN ACETABULUM TO FORM A NEW JOINT

HISTORY

Patient, male, aged forty-six, enters hospital on account of dull, throbbing pain in left hip, and also a pronounced diminution in size and marked shortening of left leg. Present trouble dates back forty-three years, when he was three years old. He was awakened one night with excruciating pain in left hip, and cried out with agony. Pain lasted only a few minutes, and he slept the remainder of the night. A few days later an extension was put on left leg and continued for a few weeks. Then a plaster cast was applied from toes to abdomen. Had no pain after application of extension. Cast was kept on for eight months, and on its removal marked shortness of the leg was noted. Patient experienced no more discomfort, except from the shortening of the leg, until seven years ago, when, while walking, he was suddenly seized with an agonizing, drawing pain in left hip, lasting a few minutes. Pain was terrific, and so severe that he could not catch his breath. Since then he has had occasional shooting pains radiating from hip to knee, but the more severe pain is continuous, dull, sometimes throbbing. This pain seems to be worse at night, and has resisted every manner of treatment for seven years. The pain is equally severe whether he is sitting or walking, and the only relief obtained is by means of morphin. Has always been well otherwise. Family and venereal history negative.

COMMENTS AND OPERATION

Dr. Murphy: When was the date of the onset?

Intern: Forty-three years ago.

Dr. Murphy: Where is the pain?

Intern: Between the trochanter and the tuberosity of the ischium.

Dr. Murphy: What nerve is causing that pain?

Intern: The sciatic nerve.

Dr. Murphy: He did not tell you that. He said the pain is anterior to his sciatic nerve. The sciatic is not sensitive to pressure. The pain is over the trochanter. This is a very unfortunate case for operation—unfortunate in two ways: first, because he says he has pain all the time, which means that he has a continuous irritation of his hip; and, second, because he says he is taking 8 grains of morphin a day, which means that you can double or triple that amount. It is difficult to tell how much is morphin pain and how much is genuine pain. When you look at the skiagram from one position it looks as if he has a bony ankylosis; from the second position it looks as if he has no ankylosis. He has a certain amount of motion, which gives him the pain. The acetabulum is filled with new bone. The head and neck of the femur have disappeared completely. The trochanter is very much enlarged, club shaped, and is impinging on the bone above the acetabulum. We will clean out the acetabulum and shape the trochanter so that it will answer the purpose of a new head, place it in the acetabulum, interposing a fat and fascia flap to prevent ankylosis. That will give him a new hip-joint. He is out of commission because of the pain. If we can relieve him of his pain, he can become a producer again instead of being a burden on his friends. This leg is about 4 or 4½ inches shorter than its fellow. The muscles are atrophied, because he has never used the leg from infancy. He never walks on it. It is too short. I believe there is some motion in that hip.

With the patient lying on his right side, we make the U-shaped incision. This flap will be retracted upward so as to expose the trochanter. Next we pass a chain-saw under the muscles attached to the trochanter, and remove about two-fifths of the trochanter. When this is retracted upward, the joint is in view. There is an abscess on the anterior surface of

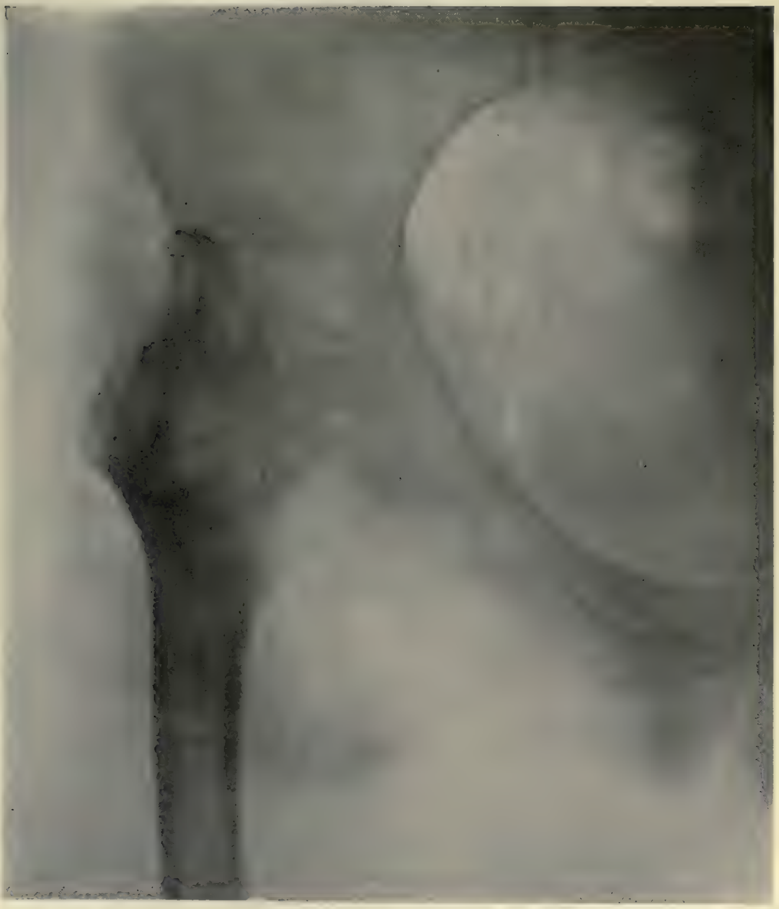


Fig. 89.—The acetabulum is filled with bone. The head and neck of the femur have disappeared completely. The trochanter is enlarged, club-shaped, and impinges on the bone above the acetabulum.

the trochanter, at the point where the neck of the bone ought to be. This is unfortunate, as in all cases in which we found pus we have had trouble with the healing. That accounts for the pain. By making traction on the leg and adducting it away across the middle line of the body the acetabulum becomes easily accessible. There is a fragment of bone firmly attached to the upper rim of the acetabulum, and impinging against the trochanter. We will remove this piece of bone with the chisel. The bone in the acetabulum will also be chisled out, and a new acetabulum made with our special reamer. This is fine! This is a much better result than anything I thought I could get. See what a true acetabulum we have here now! The upper end of the trochanter, *i. e.*, portion of the shaft on the neck side, will next be rounded off and shaped with the end mill so as to fit the acetabulum, and then we are ready to interpose the trochanteric fat and fascia flap, which will be dissected free from the under surface of the original skin-flap. The base of the flap is above. This pedicled flap covers over the acetabulum and will be sutured to its margin with phosphor-bronze wire. We have a splendid interposing flap, with a good circulation, insuring its continued vitality. That will give him a splendid joint, and were it not for the presence of pus, we could predict a good result with positiveness.

The trochanter will now be nailed back in place, and the cut edges of the periosteum at its base approximated and sutured with phosphor-bronze wire.

There was a fibrous ankylosis only, fixing the femur in an angular manner to the ilium. It was at this point that there was pressure of the trochanter against the enlarged margin of the acetabulum. Nothing remained of the acetabulum except its elevated and thickened margin. The cavity was filled in with bone. The skin will be sutured with horsehair.

Let the record show that there was an abscess on the neck of the trochanter; on the upper margin of the acetabulum was a spicule of bone which stuck out into the trochanter. This was freed. This was a great source of irritation. The upper half of the old trochanter and the upper half of the neck were chiseled off on a level with the shaft. When this was completed, we were able to throw the remaining portion of shaft out of its contact with the ilium. A new cavity was then chiseled out of the ilium with the curved chisel, resembling the former acetabulum. It was smoothed out with the reamer. Then the end mill was used on the end of the shaft, and this was rounded off so that it resembled fairly well the normal head, except that it was directly above the shaft, with no neck. This rested again in a fair acetabulum. An interposing flap was then prepared from the trochanteric fat and fascia. It was in excellent condition, with a good blood-supply. This was stretched over the new acetabulum and secured with phosphor-bronze wire all around its margin to the remainder of the former capsule, so that the new head will rest on this flap. After this was accomplished the trochanter was nailed back in the usual way. By adducting the leg we were able to rupture the adductor tendon close to the symphysis, as was indicated by the sinking in of the skin at this point. Both legs will be placed in a travois splint in abduction, and a 20-pound Buck's extension will be put on the limb. The usual dressing of the wound was made—bismuth subiodid powder, collodion gauze, 5 per cent. moist phenol gauze, and a sterile gauze dressing held in place with a roller bandage and adhesive straps. [As was feared at the time of operation, he had suppuration and discharge from the wound for weeks. He was intensely sick. Now the process of repair is advancing. He is regaining strength. There is still some pus discharge, but he has quite good motion in the hip and the leg is in good position.—Ed.]

POTT'S DISEASE

The Operation of Bone-grafting for its Cure, as Devised by Dr. F. H. Albee, of New York City

A TALK BY DR. F. H. ALBEE AT THE CLINIC AT MERCY HOSPITAL ON FEBRUARY 19, 1913

DR. MURPHY: Gentlemen: We are honored by having with us today a man who is the first to make orthopedic surgery not a borderline surgery, but a part of real surgery. He has struck out into a new line, and that is what makes him a genius. That he has succeeded, his operations show. I have the pleasure of introducing to you Dr. Albee, of New York.

DR. ALBEE: I do not know as I shall be able to say hardly a word after such a flattering introduction by Dr. Murphy. In fact, it was only after some hesitancy that I agreed to say anything.

Continuing along the line of Dr. Murphy's remarks, I might add a few words relative to the work in which I have been engaged. In the first place, you all know, as I do, that Dr. Murphy has no equal in the work of bone-grafting. He has given bone-grafting in surgery the greatest impetus which it has had in many years. I agree with him in his teaching as applied to my branch of surgery, in which bone-grafting has a wide application. The field of the bone-graft in orthopedic surgery I am sure is destined to enlarge far beyond the application to which we have had the privilege of using it thus far.

It might be interesting to mention some facts relative to the 104 cases of Pott's disease of the spine on which I have operated in the last twenty-two months, and in which I have used bone-grafts. Some of these cases had a psoas abscess and some paraplegia. That Pott's disease is amenable to treatment by bone-grafting is shown by the excellent results obtained in these cases. In every case of paraplegia the paralysis has cleared up, the time varying from three to five months.

Now, what are the premises on which we plan this operation? What is necessary in the treatment of Pott's disease of the spine? We know that mobility of the vertebræ infected with tuberculosis keeps up the process, and this applies particularly to the dorsal spine, owing to respiration. It is difficult to say precisely from what influences artificial pneumothorax derives its beneficial results. Rest and immobilization of the diseased lung tissue is the most important part of the treatment. It is certainly true in tuberculosis of the joint that perfect fixation with fresh-air treatment is as near as we can come to a specific. We have been encouraged in this by the report of Ely, which has been worked out from his pathologic studies. He says that it is only necessary to secure a bony fixation in joints in order to get a rapid disappearance of the tuberculous process. The next thing necessary is to prevent the crushing of the vertebral bodies, of course assuming that the tuberculous process occurs only in the vertebral bodies. This crushing of the cancellous structure not only favors extension of the disease, but produces an increase of the kyphotic deformity. Yesterday I saw at the hospital a man who had Pott's disease for forty-one years—since he was four years old. It had spread from one vertebra to another until nearly the whole dorsal spine had become involved. You can, therefore, see the value of this bone-grafting when you compare the relatively greater value of a bony ankylosis with a fibrous ankylosis.

One of the inspiring things about bone-grafting is its possibilities. In a letter I have just received from Dr. Twinch, of Newark, N. J., who assisted me in a case that had been under conservative treatment for seventeen years, and who had developed a pronounced kyphosis and paraplegia, he writes that the patient is now able to walk—ten weeks after the operation. In that case we split the spinous processes and bent in the bone-graft.

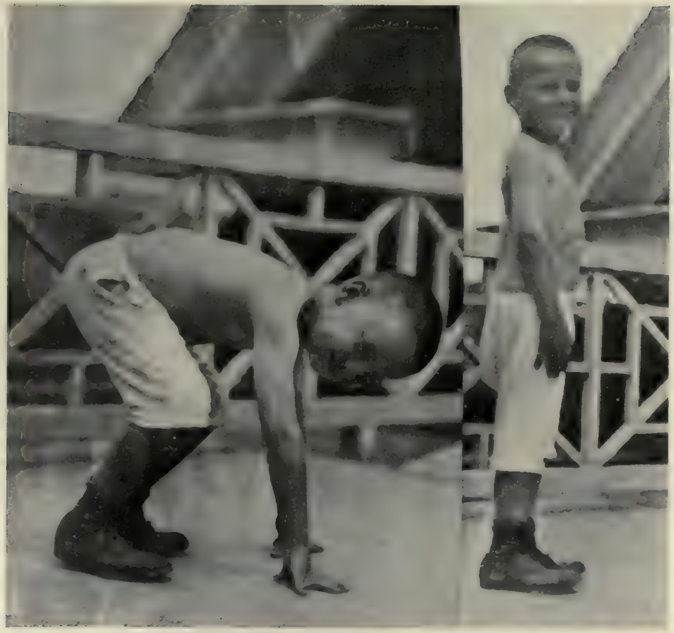


Fig. 90.—This boy, a very acute case of Pott's disease, was operated on at the Post-Graduate Hospital, New York City, over twenty months ago. No plaster jacket or brace was used. These photographs show him as he is now, and the flexibility of his spine above and below the graft.

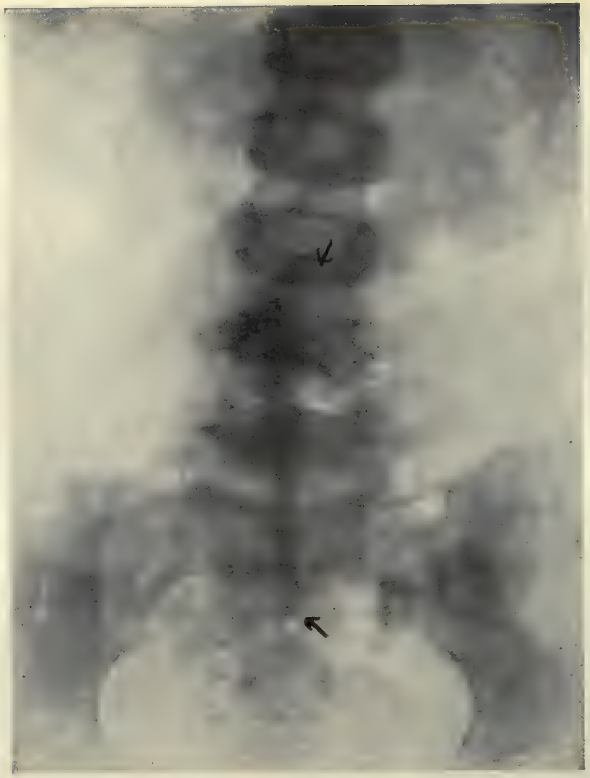


Fig. 91.—*X*-ray taken six months after operation. Case of acute Pott's disease. Child was kept on gas-pipe frame for seven weeks. Symptoms were immediately relieved after operation, and have remained so. Child runs about and plays like normal children.

Fig. 92.—Anteroposterior *x*-ray taken eighteen months after bone transplantation operation. The graft, as well as the vertebral bodies, has increased in density since the operation.

In the first 50 cases the bone-graft was removed from the tibia by means of a chisel and mallet.

We now remove the bone-graft, usually by means of the motor saw, which we find shortens the time of operation materially, increases its accuracy, and furnishes a section of bone of a more adaptable shape and size to fit into the groove formed by the split spinous processes. Our main object is to splint the diseased area, but we can also at the same time straighten the kyphosis appreciably. In cases of long duration we probably do not correct the curvature of the spine so much as we produce a bony fixation. In some of our cases we have succeeded in making a complete correction of the kyphosis. In others we have secured a small amount of correction of the kyphosis by previous fixation on the gas-pipe frame before inserting the bone-graft.

One of our original uses of the bone-graft was in cases of deformity of the lower extremities. In certain congenital club-feet in children over three years of age, where adduction of the fore part of the foot is a prominent feature of the deformity, a wedge-shaped bone-graft taken from the upper end of the tibia was inserted into the scaphoid bone. We have also taken out a wedge from the cuboid in cases where that bone is much hypertrophied and oblique in its position, and forced it in between the separated halves of the split scaphoid bone (on the inner side of the foot), thus propping the foot into a corrected position. A relapse is not likely to occur, and the continued use of braces is unnecessary, as is often the case after the Phelps operation, or other soft tissue operations where we have to hold the foot corrected by means of a brace.

The bone-graft has been used to advantage in ununited fractures of long standing in long bones. In such cases we have inlaid into the cortex of the fragments a bone-graft taken from the tibia, of sufficient length to bridge the fracture and sclerosed portion of the ends of the fragments, and reach well into the normal bone structure in both fragments, just as a cabinet-maker inlays a piece of wood. This method has been used with 100 per cent. of successes in 12 cases, and it is believed to be preferable to Codivilla's periosteal bridges.

Dr. MURPHY: I am pleased that Dr. Albee has laid so much stress on mobility as the etiologic factor in the continuance of the tuberculous process. That is a very important thing. I was glad to have him mention the immobilization of the lung by artificial pneumothorax in connection with the prevention of the spread of the tuberculous process in the lung. You will remember that in 1898 I read a paper on that subject before the American Medical Association at Denver, and predicted that it was to be the future treatment of tuberculosis of the lung. It was not taken up by my American colleagues, and since that time the treatment of pulmonary tuberculosis has gone out of the domain of surgery into that of medicine. Dr. Lemke carried out this work for a long period of time before his death. Since that time the work has been done in only desultory manner. Fortunately, the Germans took it up recently, and now they are vying with each other to see who can do this operation the most frequently. After the method has received the proper German baptism, it will come back to America and be used here, because the principles are right and principles are eternal.

There is no type of treatment that is as important as the method of immobilization in treating lesions of the spine, as was just mentioned by Dr. Albee.

REMARKS BY DR. J. B. MURPHY IN DISCUSSING THE PAPER READ BY DR. F. H. ALBEE AT THE MEETING OF THE CHICAGO MEDICAL SOCIETY, ON THE EVENING OF FEBRUARY 19, 1913

Mr. President and Members of the Society: I have been very much interested in Dr. Albee's paper. I concur in all the things that he says concerning bone-grafting, and I am delighted that the orthopedists have finally come to a renaissance and have gone into real active work in our branch of surgery.

The question that is not settled in this work on diseases of the spine is, What will be the final outcome of the case? This method of bone-transplantation for Pott's disease has not been in practice long enough to allow us to draw definite conclusions. When the spinous processes have been fixed for a time, will the patient develop another kyphosis as the result of his growth?

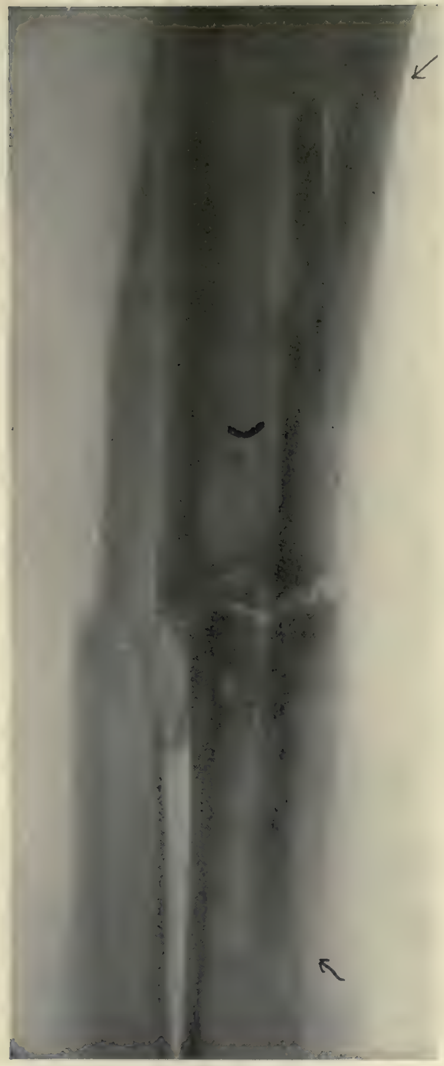


Fig. 93.—Ununited fracture of the tibia and fibula of ten and one-half months' non-union. Graft five inches long taken from crest of his other tibia and inlaid according to technic described elsewhere. Leg was firm five weeks after operation, and has remained so—now eighteen months. This skiagram was taken three and one-half months after operation, and, as will appear, there has been practically no osteogenesis from the ends of fragments themselves; the union has depended upon osteogenesis from the graft.

We undertook to demonstrate that last summer during my vacation. Dr. Zapffe carried on the experiments. He took one subperiosteal graft from the tibia and fixed it to the tips of the spinous processes in a three-weeks'-old pup. In a second experiment he transplanted a similar graft onto the bodies of the vertebræ extraperitoneally. What happened as the pups grew? The first died one month after the operation. The last dog on which the experiment was tried died five months after the operation, and through the bungling of the man in charge the dog was burnt, so that we have no permanent record of the experiments. However, what I wanted to bring out was that as the last pup grew it developed a most pronounced kyphosis. He looked like a young camel. Why that should happen we could not tell. It is generally believed that a transplant does not grow in length. Bones grow in length only from the epiphysis. If the graft is fixed to the anterior surface of the body of the vertebra, as the posterior portion of the vertebral body grows, does it tend to develop a kyphosis? In the case of children, where the spinous processes have been fixed, according to Dr. Albee's method, will that fixation be permanent? The doctor says it will and we believe it will. If not, what must happen? A kyphosis will be developed.

Now, then, we come to the essential point in bone transplantation. The limit of extent of bone transplantation is settled. That the transplant needs or does not need periosteum is not definitely settled, but that does not matter. We do not care as surgeons. We can always remove the periosteum with the transplant because it is always available. That you can transplant, as brought out by Dr. Albee, any length of bone is true. I had a case of osteomyelitis of the tibia recently in which only a small button of the internal malleolus was left, and about four inches of tibia above. I put in a ten-inch transplant removed from the opposite tibia, contacting it with the portion of malleolus below, and we already have a good result. All that is needed is contact with living osteogenetic bone, no matter how small the contacting surface may be, and any size transplant that can be obtained will live, provided it remains aseptic.

I was very much pleased with the statement made by Dr. Albee about asepsis. I have had 100 per cent. of successful cases of bone transplanting to date. Until yesterday I thought that I was going to have a failure. I transplanted a piece of bone into an osteomyelitic tibia some three or four weeks ago in the case of a boy who was in the second week of an attack of grip. We believed that he had recovered, or we would not have operated. He got a hematogenous infection around the transplant, and at one time I thought I would have to remove the transplant. Yesterday, I cut down on it, exposed it to view, and to my great delight found the graft was living.

Whether the transplant has a medulla or a cortex does not matter at all. We have had cases in which the graft had no medulla. In one case, which I reported, the transplant contacted with living osteogenetic bone at only one end. I had another case in which I transplanted 5½ inches of the tibia into the upper end of the femur, which had been removed because of a sarcoma. We replaced the muscle attachments in approximately their normal position, fixing them to the transplant with a wire nail. In putting in the nail I fractured the transplant. I could not remove another piece of bone from the tibia from which the first fragment was taken, and I could not remove a piece from the leg on which I was working, so I simply wired the fragments of the transplant together. The upper fragment was separated entirely from living bone. It was not contacting with living bone—merely with the lower fragment of the transplant. The skiagram showed later that not only was regeneration going on through both fragments of the transplant, but a fairly good trochanter was developed.

I am pleased to have the orthopedists take the stand that there is no limitation to the use of a bone-transplant; that these fragments of bone can be put into the spinous process, as Dr. Albee has shown, and live. This method is the final step in the repair of tuberculous disease, and I am sure that is what Dr. Albee is trying to bring out tonight. What the final results will be, time alone can determine. Tuberculosis will not disappear in a week or in four or five weeks. Any man who has made a study of the pathology and repair of tuberculosis knows that that is true. Whether the fixation which is produced by this bone splint which fixes and immobilizes the spine will favor the rapid and permanent cure of the tuberculosis, we are unable to say. We have always believed that no matter whether it be tuberculosis of the lung or tuberculosis of the bone, that perfect immobilization will give the very best aid to the process of cicatrization. It is the formation of connective tissue around the tuberculous process—an encapsulation that prevents the spread of the process.

CLINIC AT ST. LUKE'S HOSPITAL, CHICAGO

BY DR. FRED H. ALBEE, OF NEW YORK CITY, ON FEBRUARY 20, 1913

Dr. Albee: I do not wish to say much, for fear of repetition, but there are a few things which I think were not brought out clearly enough last evening. In the first place, I would like to make a little clearer the rationale of this operation. The object is to produce a bony fixation and to prevent the irritation of motion and crushing of the vertebral bodies, and thus stop the progress of the kyphotic deformity and the tuberculous process. Nature is not efficient in combating tubercular joint disease, because of the slow and meager osteogenesis which takes place in an active tuberculous joint; thus respiratory motion and vertebral crushing from superincumbent body weight and muscular spasm are unlikely to be presented, and the progress of the disease is unabated.

The success which attended my operation for ankylosing tubercular ostitis and osteo-arthritis of the hips during the past six years in numerous cases led me to endeavor to devise some method which would apply equally well to tuberculosis of the spine.

I believe there are few cases of tubercular ostitis of the knee in which an erasion has been performed with a resultant bony ankylosis, in which the tuberculous process has not subsided rapidly. It is very satisfactory to produce a bony stiffening of the tuberculous knee, and still better of the tuberculous hip, because in the latter instance there is compensatory motion at the lumbar spine, which is in a plane only approximately 2½ inches higher than the hip-joint.

The operation which we have been doing for some time for tuberculosis of the knee has not for its object the removal of all the diseased tissue. The aim has been to remove as little of the bony tissue as possible for the purpose of fixation. When ankylosis is secured, the diseased area is fixed and the patient is benefited by the cessation of symptoms and the rapid arrest of the disease. This also holds true in the application of the bone-graft to fix the diseased vertebræ of the spine.

The spine is anatomically well constructed for this method. We are able, by placing the bone-graft in the tips of the spinous processes of the diseased vertebræ and one or two normal ones, above and below, to make use of the full length of the spinous processes as levers over the transverse processes as fulcra, and thus hold that segment of the spine in hyperextension. This prevents motion at and crushing of the diseased vertebral bodies, both by leverage action of the spinous processes and splint action of the graft, and the field of operation is superficial, posterior to the spinal cord, and distal to the focus of disease.

CASE I

History.—Patient, a male, aged six, was brought to hospital because of a deformity to spine. The patient is very weak, frail, and anemic, extremely slender, and appears markedly tubercular. The entire chest is deformed, due to a long-standing kyphosis opposite the seventh to twelfth dorsal vertebræ. There is a marked dorsal curvature, producing quite a prominent lump over the tenth dorsal spine. The skin appears reddened and is very tender to touch. There is no associated scoliosis. Associated with the deformity there is a moderate degree of muscular rigidity which limits the bending movements at the back. The parts are painful to palpation, especially in and around the kyphosis. There is no associated paraplegia or evidence of any abscess formation.

Past History.—Has had none of the diseases of childhood. Never had a large appetite. Has never had toothache.

Family History.—Father and mother both living and well. Has two brothers and two sisters, all living and well.

OPERATION.—I am making the skin incision, beginning at the midline above, swerving to one side, and then back to the midline below the diseased area. This circular flap I will dissect up to expose the spinous processes with their supraspinous ligament, which I now split directly over the tips of the spines. The scalpel often tends to slip to one side, but this has to be prevented. I believe it better to make the curved skin incision to the side, as subsequent pressure and infected sutures would not come over the wound, and also better protection afforded the graft.

With the scalpel I split the supraspinous ligament at the same time that I split the interspinous ligaments between the tips of the spinous processes. Notice I am not separating them from their attachments to the spines. I am splitting them *en masse*. With the chisel I am now splitting the spinous processes themselves, and am breaking the halves at the bottom of the incision on this side, being careful not to fracture the half on the opposite side.

In splitting the spines we are guided by the sound of mallet and chisel as we approach the neural arch, which has proved to be a safe guide to prevent the chisel from penetrating too deeply. I have never had the misfortune to enter the neural canal

VISITING DOCTOR: You will not be able to correct that deformity to the full extent?

DR. ALBEE: No; it is a kyphosis of too long duration.

DR. PORTER: It is three years since the trouble first started.

DR. ALBEE: On account of the nature of this curve, we will have to bend the graft in putting it in. I would rather have put it in without bending it. I would like to call your attention to the motion between the vertebræ making up the kyphosis: I think I can see motion right there in the center of that kyphosis, and in view of this motion it is evident that if we put in a bone-graft as a splint and fix it in the spines under lateral tension, a certain amount of kyphotic recession will result. We take a measure with this flexible probe of how long the graft is to be, in order to span the diseased vertebræ and one normal vertebra above and below. Now we outline on the exposed tibia what is to be the transplant, by incising deeply the periosteum where the saw is to travel. We will have the saline solution drip on the saw to keep it from heating. With the motor saw we remove a splint of bone from the crest of the tibia with a cross-section, thus [indicating].

Visiting Doctor: Do you leave the periosteum on your graft?

Dr. Albee: Always, and some marrow tissue also. In my dog experiments, as I said last evening, I have used a large number of grafts without the periosteum, and these grafts seemed to do equally well as did those with the periosteum, but as the question about the permanency of the graft without the periosteum has been raised, I always include the periosteum when operating.

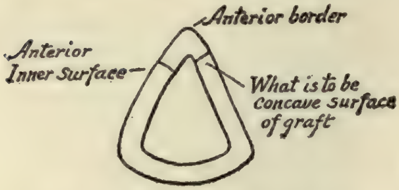


Fig. 94.—Diagram showing manner of taking the graft from the crest of the tibia.

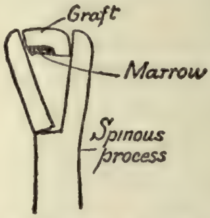


Fig. 95.—Diagram showing graft in place. It is held there by the supraspinous and interspinous ligaments being pulled over it posteriorly by the kangaroo-tendon sutures.

We will have to bend this graft in. It will be impossible to get the graft wide enough to mold it into the segment of a circle. Now we will have the saline solution drip on the saw.

With the graft held in two broad ligament clamps we are making with the motor saw notches one-half to two-thirds the way through the splint, on what is to be its concave side (see drawing). We are imitating the carpenter in this matter, as he does precisely this when he wishes to bend a board around a corner. We have no trouble in bending children's bones under these conditions.

Now we will cut off the tips of the graft slantingly, so as to leave no sharp corners to project under the skin. I will incise the periosteum on the graft obliquely, so as to improve the blood-supply to the graft, and let out those active osteogenetic cells from the periphery of the bone-cortex.

Kangaroo tendon (medium) is a good suture for this work, because it is very strong. You need something hard and strong, because when I get down to this end, there is considerable tension, and catgut breaks. See! there is a good deal of tension in the center.

I think that we can expect some correction, because we found motion as the child breathed. We did not need the leverage of these two middle processes at all—just the leverage of the spinous processes on the side. Therefore we have cut off their tips with bone forceps, so as to diminish the kyphosis still further.

The next case is not very much deformed, and I will demonstrate putting in the graft in another way. We will put it in on the edge. It will have more strength if put in on the edge.

[Sutures, applies dressing and adhesive straps to hold dressing.]

In a case where the kyphosis is marked it is most important to build up about the knuckle with gauze or cotton pads, so as to prevent pressure on the tip of the kyphosis, because of the danger of a pressure-sore.

VISITING DOCTOR: What is the after-treatment?

DR. ALBEE: Keep them on a gas-pipe frame or mattress with board under it (fracture-bed) for six to ten weeks.

VISITING DOCTOR: Do you put on a cast?

DR. ALBEE: I have not used a cast on a single case: I rely on the graft.

VISITING DOCTOR: Do adults find the board hard to lie on?

DR. ALBEE: In our older cases we use a mattress with a board under it entirely. In one case of an adult where I used a frame the patient developed a pressure-sore over and down to the graft, but it healed in completely and the graft is as good as ever. The knuckle was very sharp in this case.

VISITING DOCTOR: Do you keep them on their backs on that mattress?

DR. ALBEE: Yes, from six to eight weeks.

VISITING DOCTOR: Do you suppose that getting the patient on the bed-pan ever injures the graft?

DR. ALBEE: No. I have a very thin bed-pan used, but we are planning to get a sectional bed and thus be able to take out a section of it, instead of raising the patient.

A point about the technic where the child has been wearing a plaster jacket: A plaster jacket produces a pressure atrophy of the spinous processes in the tip of the kyphosis in nearly all these cases. In these cases the graft is very necessary if a bony bridge is to be produced by operative precedure, because of the small amount of bone present and the shortness of the spinous processes. Also we would caution against going too deeply with the chisel, and thus entering the spinal canal.

VISITING DOCTOR: What has been your experience with grafts of other species?

DR. ALBEE: Experiments have been done in which grafts of sheep's bone were inserted into dogs and they were all unsuccessful. I believe that if we are going to develop surgical procedure for human cases, we should use the most reliable methods and material, and I think an autogenous graft is the most reliable. I think grafts from other animals, dogs or sheep, are very unreliable. I have used successfully grafts taken from children strangulated at birth, which grafts have been kept in cold storage for forty-eight hours. I have also used a large part of the astragalus taken from a young adult, which was kept in cold storage twenty-four hours, to take the place of an absent head and neck of the femur in a child of four years. The graft was fastened to the stump of the neck of the femur by a dowel made from the tibia of another individual of about twenty-six years. The upper end of the femur in this case had been destroyed several months previously by an abscess; the acetabulum had remained undestroyed—hence the amount of motion. That case has been

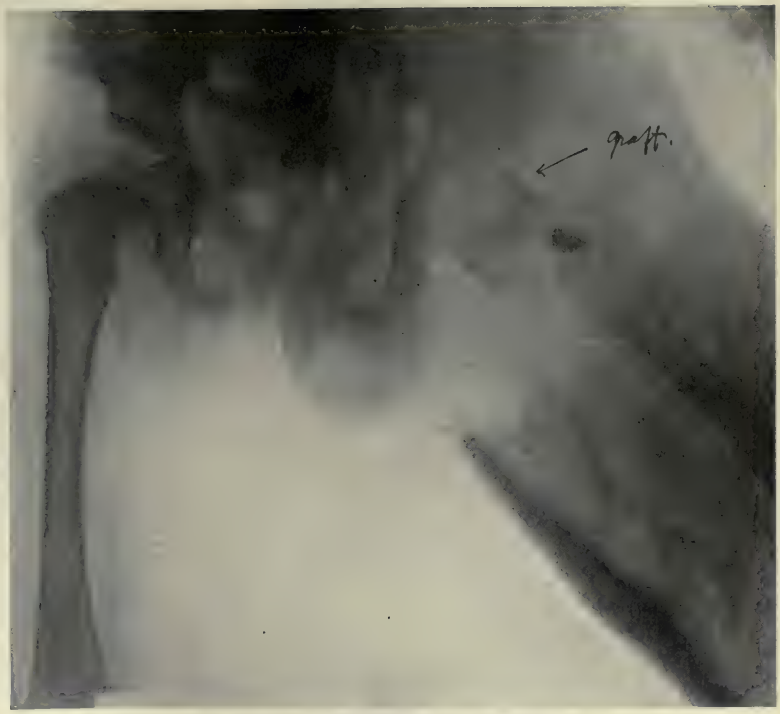


Fig. 96.—Child four years old. Destruction of head and neck of femur by septic arthritis several months before. Transplantation of two-thirds of astragalus of a young woman of eighteen years to take place of absent bone. This was fixed to upper end of femur by dowel made from piece of tibia of a person of twenty-six years.

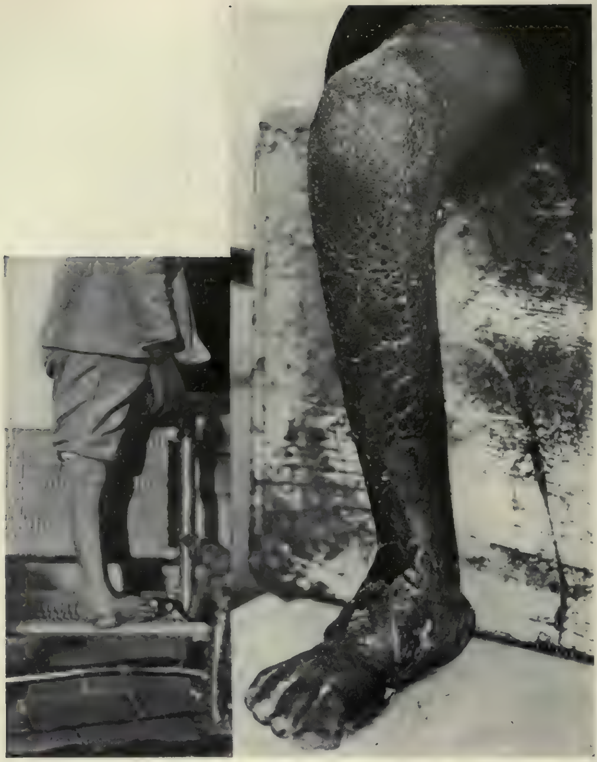


Fig. 97.—Photographs before and after a bone transplant wedge was placed into scaphoid, after splitting it into anterior and posterior halves. The graft was obtained from upper end of the tibia of the other leg.

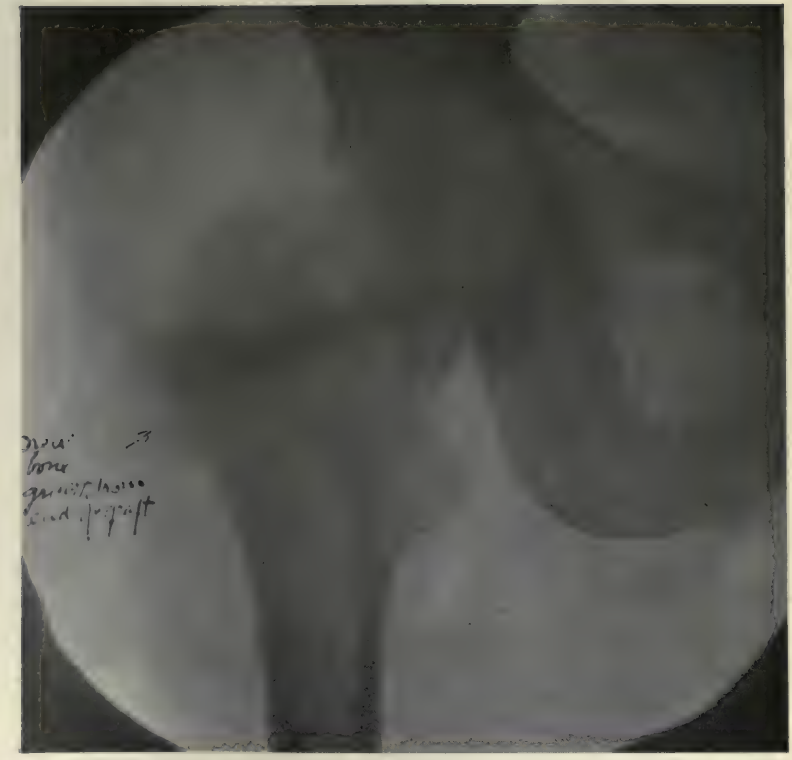


Fig. 98.—*X*-ray taken five weeks after operation. Case of ununited fracture of neck of femur of three and one-half months' duration, in woman of sixty years. In five weeks there was firm union. The most interesting feature about this *x*-ray is the large growth of bone from the external end of the graft itself, demonstrating the early bone-forming ability of the bone graft. After freshening the ends of the fragments, a hole the size of one's forefinger in diameter was made by a motor-driven drill through the outside of trochanter, through neck and head. Into this a dowel-shaped graft from the patient's tibia was driven with considerable force into place.

a most satisfactory one. The child has from one-half to two-thirds of the normal motion at the hip. These grafts are kept in cold storage at a temperature of 5° or 6° C. in sterile vaselin. Five or 6° C. is the best temperature, because the bone is damaged more by contraction and expansion if kept at a lower temperature.

We put in a bone-graft in a case of wrist-drop which has been very successful. It resulted from a paralysis. The child was six or seven years old, and every time she tried to use the arm the wrist dropped because of a paralysis of the extensor tendons. She was quite a talented young child, and tried to pick out notes on the piano. Her parents were very anxious that she should use the arm, and consulted me. The graft had never before, as far as I know, been used for that purpose, but I suggested trying it. They concurred. We put in a graft taken from the tibia of the same patient, and inserted it into the side of the radius, about two inches from the articulation of the wrist, and fixed it to the trapezium and trapezoid bones and to the ligaments. The result is most gratifying. Now, of course, we can do something for a case of this kind by applying a brace, but it would always interfere with the use of the hand.

We have also used similar procedure for paralytic conditions of the peroneal muscles in older children, where the foot is held markedly in an adducted position. A wedge-shaped piece of bone is placed between the astragalus and the scaphoid after the cartilage had been removed from each of these bones. In this way the foot is held in the corrected position. For congenital club-foot we split the scaphoid vertically and put the wedge-shaped graft in between. This is done in children over three years of age only.

CASE II

HISTORY.—Patient, a female, aged six, brought to hospital for correction of a long-standing Pott's disease. Parents state that since the child was one year old she has always had a swelling over the middle of the dorsal spine. Condition has been gradually getting worse during the last five years. Complains of pain and tenderness over back.

Physical Findings.—Healthy appearing child, well, seems to be developed in every way. Dorsal curvature of moderate degree, involving the tenth to twelfth dorsal vertebræ. Slight pain and tenderness over back. Past and family history negative.

Dr. Albee: Now, again, I split the spinous processes. Here is a child lying on this firm operating table, and remember that she is breathing a little more forcibly than normal, and there is all that motion between the vertebræ of the kyphosis. See how much we can increase the motion by moderate pressure over the kyphosis.

In this case the spinous processes are very much shortened from the jacket which the child has been wearing. I cannot go in there as deeply as I would like to, on account of shortened spinous processes, so I will have to bend this graft.

[Takes piece from tibia with motor saw and saws notches into graft by precisely the same technic as in first case.]

I find it well in adults, where we do not wish the graft to grow in length, to incise the periosteum longitudinally. In the case of children, where we want the graft to grow, we incise it crosswise, following Macewen's teaching, that the periosteum is a limiting membrane. Now you see that graft goes in very nicely. We have not used many hemostats. There is very little bleeding, and this is the usual experience. We have not cut any vascular structures.

Visiting Doctor: Do the grafts ever break?

Dr. Albee: This is my one hundred and eighth case; if this does not break, I will have had only one in that number that broke. That case was an adult, a medical student. The result has not been interfered with thus far, as he declares that the pain disappeared when he went under the ether and has not returned since the operation. The periosteum was not torn where the graft broke.

You notice the large amount of motion right in the center of the kyphosis in a case of this duration. Here is a case of five years' duration. There is motion in the center of that kyphosis each time the child breathes. I never noticed that amount of motion in my first cases. We have not tied a bleeding vessel during the whole operation. We have a very good hemostasis.

VISITING DOCTOR: How soon do you remove those dressings?

DR. ALBEE: After eight or ten days.

VISITING DOCTOR: Do you ever use the gas-pipe frame?

DR. ALBEE: We generally use a fracture-bed (board under mattress), sometimes a gas-pipe or Bradford frame. The trouble with the gas-pipe frame is that the children are uncomfortable. Take a kyphosis like this: it is rather difficult to pad that so as to prevent considerable pressure on tip of kyphosis, and too much pressure might produce a pressure-sore. That is why I believe in using the fracture-bed. I keep them on their backs for six or ten weeks. I keep some of them in the hospital for three weeks, and then send them home on a frame.

PROCIDENTIA UTERI

Dr. Murphy's Method of Fixing the Uterus

HISTORY

Patient, female, married, aged fifty-six, enters hospital on account of prolapse of the uterus and a constant uterine discharge. Trouble dates back to thirty-three years ago, when her first child was born. It was a hard labor and she was badly torn. Two months later she noticed a mass coming down in her vagina. It was much worse after being on her feet a great deal or after straining at stool. Three years later her second child was born, and two years after this a third child was born. Neither of these pregnancies seemed to affect her trouble one way or another. In 1890 she was told that she had several ulcers on the cervix. They caused her much pain, and occasionally there was a slight vaginal discharge. They were treated by medication and healed. Her condition now remained about the same—no pain and no other symptoms unless she was on her feet a great deal. Her bowels, however, were very constipated. In 1905, after taking care of a sick person for six weeks and doing much lifting, her trouble began to get worse, and it has progressed steadily until the past few months, when it has grown worse rapidly. Now the uterus protrudes from the vulva three or four inches when she is on her feet. During the past three months she has had a dull aching pain through the pelvis and back. Occasionally she has slight difficulty in passing urine. Bowels have not been so constipated during the past seven years as previously. Since September 1, 1912 (five months ago), she has had a continuous vaginal discharge. It is bloody most of the time, with not much odor. It varies slightly in amount, but is always constant, requiring two or three napkins a day. Menopause three years ago. Menstrual history negative. Past history is negative except for periodic attacks of diarrhea about seven years ago lasting for five years.

COMMENTS AND OPERATION

Dr. Murphy: Several factors must be considered in the evolution of this condition. There is, first, the retroversion. Procidentia cannot occur until there is a retroversion; second, the increased intra-abdominal pressure on the anterior surface of the uterus when in the retroverted position presses the organ down until the perineum gives way, and the tampon pressure of the cervix and the wedge-shaped enlargement of the uterus do the rest; third, this state of affairs continues, getting a little worse all the time, until the patient makes a supreme exertion, and then the uterus appears at the vulva. In these cases the question arises whether one should do a hysterectomy or a ventral fixation in the manner suggested by Kelly for retroversion. This is always a good operation if it is done early, in the retroversion stage of the procidentia, thus avoiding the procidentia by putting the uterus in a position where it does not retrovert. Now, however, we must do something that will lift up the whole floor of the pelvis and hold it up permanently. If one could close up the vagina completely, approximating the levator muscles, leaving no opening, then the perineal operation would do very well, because it would throw all the intrapelvic weight on the perineal muscles, but you cannot do that in these young women with marital relations. Next you can do a supravaginal amputation and bring the broad ligaments across the stump of the cervix to hold up the vagina. After that commences to stretch, out come the rectum and the bladder, and the patient is worse off than before.

In these cases we have worked out a plan that has so far been entirely satisfactory. If we see the case in the early stage, we do a ventral implantation; that is, we implant the fundus of the uterus between the divided aponeuroses of the abdominal rectus muscles and unite it to the rectus muscle so that it becomes fixed there. We divide the tubes from the uterus, taking them

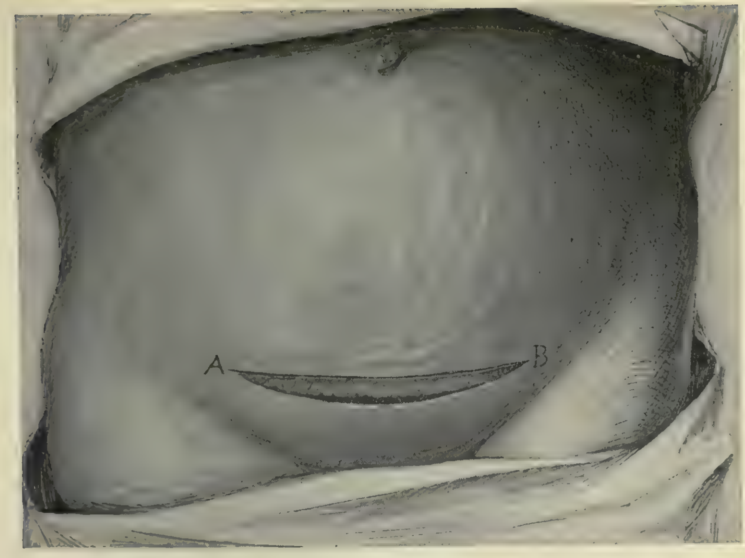


Fig. 99.—The Murphy operation for procidentia of the uterus. First step. The drawing shows the line (A–B) of the transverse semilunar incision, six inches in length, and about one inch above the symphysis pubis (Bryant and Buck, "American Practice of Surgery").

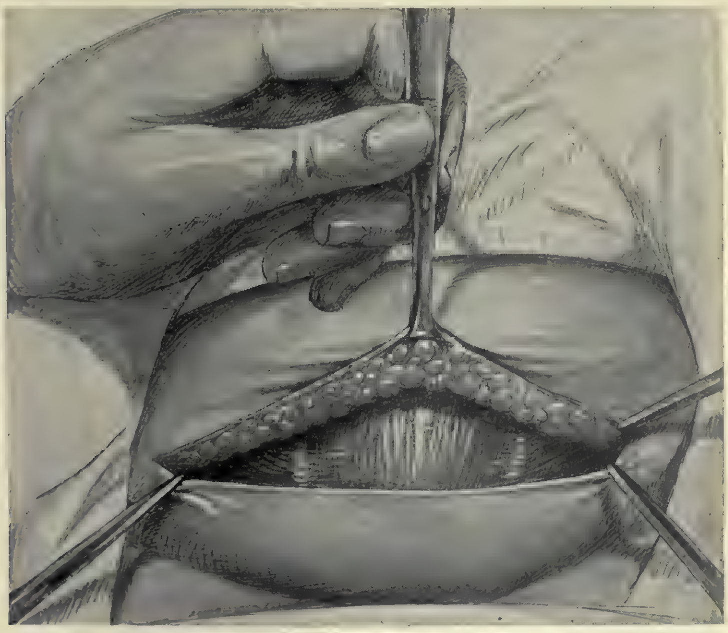


Fig. 100.—Second step. The drawing shows the skin and fat divided down to the aponeurosis of the recti. The upper flap is retracted. The fibers of the rectus muscle are visible (Bryant and Buck, "American Practice of Surgery").

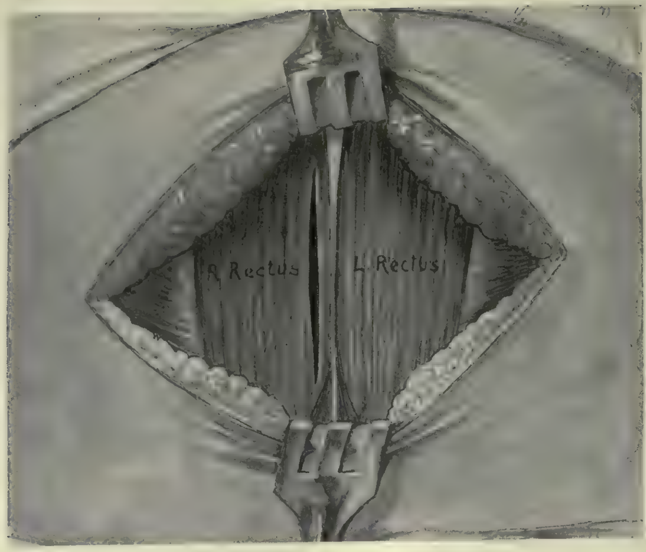


Fig. 101.—Third step. The drawing shows the line of incision in the right rectus muscle and peritoneum underneath (Bryant and Buck, "American Practice of Surgery").

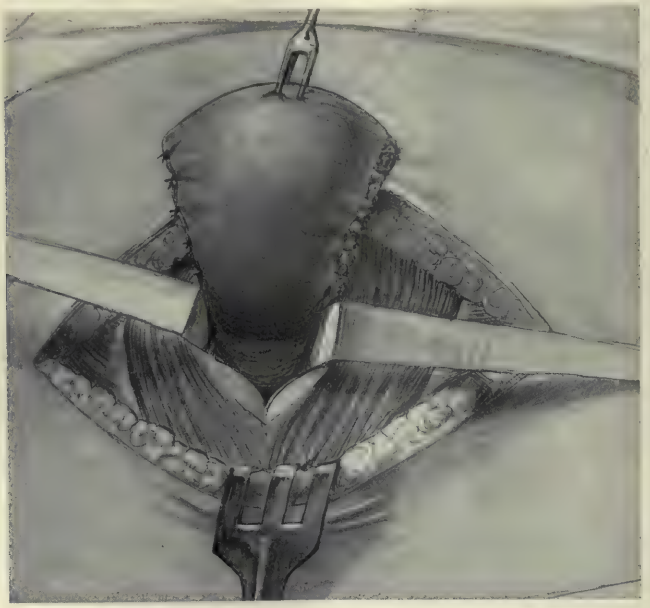


Fig. 102.—Fourth step. The drawing shows the abdomen opened and the uterus drawn out of the abdominal cavity and divested of the broad ligament on both sides down to its junction with the cervix. The lateral sutures have been placed (Bryant and Buck, "American Practice of Surgery").

off at the cornua, displace the broad ligament downward, and bring the fundus of the uterus above the anterior surface of the rectus muscle. If it is a superlative degree case, you can place more of the uterus outside of the peritoneal cavity. Often you can put the whole uterine body outside and make an anterior extraventral implantation—that is, you can draw the uterus up through the anterior abdominal wall, then split it anteroposteriorly into halves, take out the mucous membrane entirely, from the cornua to the cervicocorporeal junction. Then lay these two parts of the uterus, serous surface forward, on the rectus muscle sheaths, like the two wings of a bat, and suture them in with catgut. That holds up the floor of the pelvis for all time. You can do that operation practically extraperitoneally except the lifting of the uterus into the abdominal incision.

The technic of this operation, which was described by me originally in Bryant and Buck's "Surgery," is as follows:

The patient is placed in the Trendelenburg position. A transverse semilunar incision is made one inch above the symphysis and measuring six inches. The tissues should be divided down to the aponeuroses of the recti; the latter should be freed from fat over an area one inch wide for the full length of the incision, and the edges retracted. The right rectus is then incised for two inches close to the median line and parallel to its long axis. This incision is extended through the peritoneum. The uterine fundus is then grasped with a vulsella and brought out through the opening until the cervicocorporeal portion is clearly in view. The round and broad ligaments are then clamped with hemostats on either side down to the cervicocorporal junction and cut free from the uterus down to the tip of the forceps. The stumps are ligated, and the tips sewed accurately to the cervicocorporal junction, and this portion is slipped back into the abdominal cavity, thus leaving the body of the uterus bare and free, standing above the level of the divided recti.

The peritoneum is next sutured accurately around and to the circumference of the cervicocorporal portion of the uterus, thus closing the peritoneal cavity. The uterus is then split through the middle from before backward parallel to the long axis of the body down to the cervicocorporeal junction. It is opened out laterally, thus forming two wings. The mucosa is next cut off clear out through the divided cornua down to the cervix and removed. The two lateral flanges of uterine muscularis are then sewed firmly to the aponeurosis of the rectus all the way around, making a bat-like flange over the recti. Finally the divided edge of the aponeurosis of the rectus is tightly closed around the cervicocorporal portion of the uterus. The skin and fatty tissue are united, a small drain being left at the lower angle of the wound. The uterus can never again get back into the abdomen. The traction on the anterior vaginal wall holds the bladder in position, and the posterior vaginal wall, the rectum.

The only intra-abdominal work is the detachment of the broad ligaments. The stumps of these are covered by suturing, so that no abraded surface is left within the peritoneum at the completion of the operation. If the operation is performed before the menopause, great care should be exercised in removing all the uterine mucosa, as otherwise periodic hematomata will form at the menstrual periods. The operation can be done in twenty minutes by trained hands.

In recent times the employment of pessaries in the treatment of advanced prolapse has been superseded by operative measures, for the reason that the condition does not permit of spontaneous recovery, but tends to become aggravated with advancing years. Moreover, treatment by mechanical means, as by pessaries or tampons, is in no sense curative; it depends upon putting the vaginal tissue upon the stretch and thus supporting the uterus. It is safe, therefore, to assume that, after a number of years of such treatment, many cases will outlive the usefulness of the method. Surgical interference, on the contrary, is curative and is attended with comparatively little risk in the hands of skilled men, even though the patient be well advanced in years. Trained men frequently operate without anesthesia, or under local anesthesia, thus avoiding the risk of a general anesthetic.

Operative treatment is divided into two classes, conservative

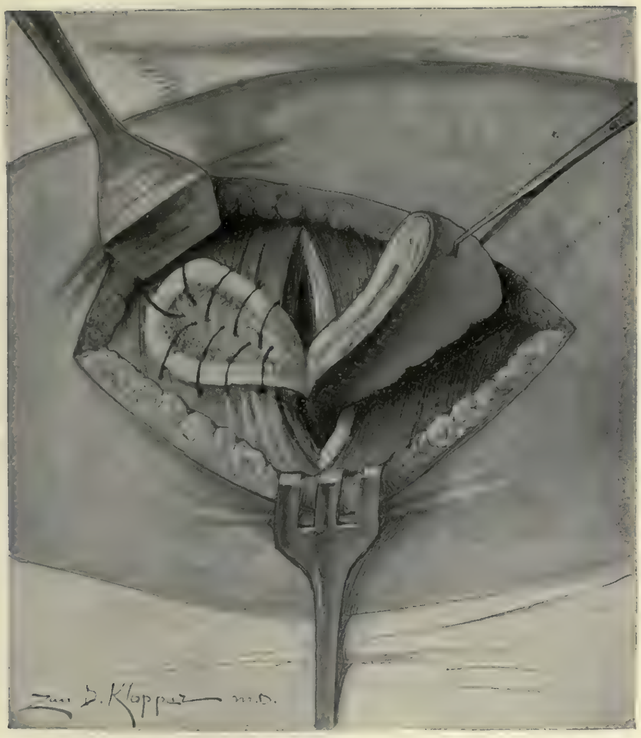


Fig. 103.—Fifth step. The drawing shows the right half of the divided uterus sutured on the anterior surface of the right rectus muscle, the left half being ready for suturing. The uterine cavity surface, which has been divested of its mucosa, is uppermost. The cervix is in the peritoneal cavity (Bryant and Buck, "American Practice of Surgery").

and radical, according as the uterus is allowed to remain or is removed. Conservative operations are applicable generally in cases of incomplete prolapse, while the radical procedures are usually reserved for complete procidentias, unless there is some indication for extirpation, such as is afforded by ulceration, etc. Both operations are supplemented by procedures which restore the pelvic floor and narrow the lumen of the vagina.

The operative treatment which up to recent times had enjoyed the greatest vogue consists in fixing the uterus to the abdominal wall after a preliminary curetage, amputation of the cervix, and ablation of the anterior and posterior vaginal walls with colporrhaphy. As we have already indicated, fixation of the uterus should not be done upon a woman capable of becoming pregnant. Therefore, as a preliminary step in the operation on women who have not passed the menopause the tubes should either be resected or a wedge-shaped portion should be removed. If this is not done, and if sutures are tied about the tubes, the lumen of the tubes may later be restored and pregnancy result. Therefore if the tubes are to remain, a wedge-shaped area should be removed from the cornua and the gap should be closed over by plastic sutures; the ligated end of the tube should be buried in the broad ligament.

The study of chronic cases has shown that the method of fixing the uterus, as performed in this country up to fifteen years ago, does not lead to good results. The fixation, as a rule, is not firm, and there is formed a suspensory ligament which later elongates. It is better, if this method is decided upon, to make upon the fundus a raw area several centimeters in diameter, and around the edge of this denuded area to fix the peritoneum with a continuous suture of catgut. Then, in closing the recti muscles, seize the scarified top of the uterus in the intermuscular suture, so that the fundus shall be firmly attached to the recti muscle. It must be admitted, however, that the remote results of this method in unselected cases are not so satisfactory as to make it the operation of choice for cases of complete prolapse. Not infrequently the operator finds, some years after the operation, that, although the uterus is well held up, the bladder is again prolapsed.

Now, I have made the Pfannenstiel incision. The aponeurosis of the rectus muscles is exposed. Next I will divide this aponeurosis and retract the muscle-fibers, without injuring or dividing them. The peritoneum is now in view, and will be picked up with two tissue forceps and divided on the incline between them. This woman is just getting the incontinence which is one of the disagreeable symptoms of the procidentia. If the uterus has been drawn down in the pelvis for a time, the bladder sags and the patient has incontinence. I can pick up the uterus easily. It comes up into the field nicely. The next thing to do is to free the attachment of the broad ligament to the cervicocorporeal junction, on both sides, and ligate the uterine arteries. We are not going to take out both ovaries, only one,—the left,—because it is senile and fibrosed. After the uterus is freed I can draw it out of the abdominal cavity. I am going to split that uterus anteroposteriorly into two equal parts. The next thing is to split that peritoneum. You see all this work has been done practically outside of the peritoneum. I am going to fix that broad-ligament stump to the cervix, so that it will be fixed there and not drop down into the peritoneal cavity and form adhesions. The next thing is to suture the peritoneum around the cervix so as to close the peritoneal cavity. That leaves the body of the uterus outside of the abdominal cavity.

The rest of the work is extraperitoneal. The next step is to approximate the cut edges of the aponeurosis of the rectus muscle around the uterus, just the same as I closed the peritoneum. We leave the uterus on the outside.

Next I split the uterus and dissect out its mucosa entirely. It is senile and dry; it scarcely bleeds at all.

Then we turn each half of the uterus outward and place the serous surface in contact with the aponeurosis of the rectus, suturing it to the aponeurosis with catgut. The uterus is now spread out on the rectus muscle like a bat with wings spread. The uterus is outside of the peritoneum all the time. One can scarcely conceive of a more simple operation. When the patient has not passed the menopause, we do the same operation, but we never remove the ovaries in those cases. In patients past the menopause this is the ideal operation. Women who have a procidentia of this degree never have children, and in those cases the operation is also indicated. It is not difficult to do. I think it is about five years since I did the first operation of this kind. If the bladder comes down, we will do an anterior colporrhaphy to correct the cystocele or bladder descensus. We have not had to do that operation in a single case up to this time. A small gutta-percha drain will be put in and the wound closed in the usual way. [The patient made an uneventful recovery.—Ed.]

CHOLECYSTITIS: SYMPTOMATIC DIABETES MELLITUS DUE TO GALL-BLADDER INFECTION

DISCUSSION OF MEDICAL PHASE BY DR. C. L. MIX

HISTORY

Patient, a female, married, age forty-three, enters the hospital because of epigastric pain and general weakness.

In July, 1912, the patient was in bed for four weeks with gaseous distention of the abdomen; she could eat scarcely anything on account of a burning sensation in the epigastrium, immediately following eating, and later great distention. She had a dull pain over the whole epigastrium, she thinks due to pressure. She had no nausea or vomiting. She had "cramp-like" pains over the abdomen which came and went with no regularity. She also had an alternating diarrhea and constipation. A doctor whom she consulted told her she had diabetes.

Previous to this, for about four years, she had great thirst and a keen appetite, with very frequent urination and a severe pruritus around the genitalia. She got up after four weeks and was very weak for some time. She lived on a diet during the whole summer of 1912, and felt very well. Last fall, when the cold weather started, she began to have pains in the epigastrium and both hypochondriac regions. These pains grew worse after taking either solid or liquid food, within ten minutes after eating. The pain was raw and dull, remained for four or five hours, and only when she began to feel hungry did the pain disappear. She never vomits or is nauseated. She complains of the worst pain in the middle of the epigastrium. There is some pain under both ribs—no more on one side than the other. She never has had a sudden acute pain in the side. Her abdomen is tender in the midline and under both costal arches. The pain used to radiate through to the back, but never to the right shoulder-blade. Formerly she had a great deal of pain over various sections of the body—head, face, and limbs. She has been in her present condition since last November, and is gradually getting weaker.

Menstrual history: She is always slightly irregular, flows three days, and has no pain. She has six children, the youngest ten years.

COMMENTS AND OPERATION

Dr. Murphy: Dr. Mix, will you kindly discuss the medical history of this case?

Dr. Mix: You have just heard the history which has been given by the intern.

This patient first consulted a physician early in July, 1912. At that time she had lost some 30 pounds in weight, though no mention is made of it in the intern's history. The physician who made the examination found, evidently, a large amount of sugar in the urine and stopped with the diagnosis of diabetes. Diabetes is not found to be associated with a burning sensation in the epigastrium and gaseous distention. Though capable of explaining very satisfactorily the general weakness and the various pains, which are mentioned in the history as formerly existing in various sections of the body, head, face, and limbs, diabetes does not suffice to explain the burning sensation and dull pain in the epigastrium. It was these symptoms which attracted our attention to the patient and for which she is this morning to be operated on.

On entrance the blood examination which was made immediately showed 75 per cent. hemoglobin and 8400 white blood-corpuscles; examination of the urine made the same day showed no albumin, no casts. Sugar was present and a few white blood-cells. The specific gravity of a single specimen sent up to the laboratory was 1027—amount, 120 c.c. A twenty-four-hour specimen collected the next day showed a specific gravity of 1035. I kept her under rather careful observation from her entrance on the fourth of February until the ninth, upon which day I found that she showed a temperature of 99° F. at 6 A. M.; at 3 P. M. a temperature of 99.4° F. On the tenth she had a temperature of 98.8° F. at 3 P. M. A blood examination made on the ninth, the day on which she showed a slight amount of fever, gave a leukocyte count of 11,800, indicating some slight infection. The following day, when the temperature had fallen to 98.8° F., the blood examination showed 8200 leukocytes. From this I felt confirmed in the opinion that some infectious process was located somewhere in the body, and was probably associated with the epigastric pain of which she complained.

The examination of the urine made yesterday showed a specific gravity of 1043, and that reported this morning shows a specific gravity of 1040, with a percentage of sugar of 6.9. As to the diagnosis of diabetes, therefore, in this case there is absolutely no question; in looking over the history you will recall that for four years she had been troubled with a great thirst and appetite, frequent urination, and severe pruritus around the genitalia.

In July, 1912, there is a definite history of some abdominal disturbance. She was in bed for four weeks with a loss of appetite and a burning sensation in the epigastrium, together with dull pain, gaseous distention, some alternating diarrhea and constipation. At this time there was no nausea or vomiting. She also believes that during July, 1912, she had fever. After remaining in bed for four weeks she recovered sufficiently to get up and go about her work, but in the fall of the year, with the onset of cold weather, she again became troubled with pain in the epigastrium and under the edges of both the right and left ribs. Both in July and in the fall she said that the pain was quite severe in both hypochondriac regions.

At the onset of cold weather she began to notice some relationship between food and pain. The pain became worse about ten minutes after eating, was raw and dull, lasted four to five hours, and usually did not disappear until she began to feel a sensation of hunger for the next meal. To me she complained of an additional symptom which she described as cramps in the abdomen when she walked, but I found on cross-examination that she meant merely by the word cramp a pain. She never was jaundiced; she never had a chill; she never had any pain in the right iliac fossa.

Physical examination shows tenderness in the midline and under the right costal border. The amount of tenderness on deep pressure was rather considerable, and suggests gall-bladder disease. Feeling that there might possibly be a gall-bladder infection from typhoid germs, though there was no typhoid fever mentioned in her life history, I ordered a Widal reaction, which proved to be negative.

The history of the case might suggest a duodenal ulcer or an infected gall-bladder. Examinations of the feces for blood were made three times, and in each case the report was negative. Evidently if there is an ulcer in the duodenum it is at the present time quiescent. The pain which was mentioned as occurring within ten minutes after eating is not at all suggestive of a duodenal ulcer. We have been accustomed to find individuals with duodenal ulcer complaining of pain from one to three hours or more after meals, and continuing until the next meal has been eaten. In the present case we note the occurrence of pain ten minutes after meals, and its disappearance, not when the patient had eaten, but when the patient began to feel the natural appetite for the succeeding meal. Such a pain might perhaps be associated with gastric ulcer, but only in the event that food retention was present for several hours after a meal. Examination of the patient's food progress through the stomach showed no delay whatever, and a normal stomach digestion with normal acidity.

On the other hand, the symptomatology is fairly well explained by the diagnosis of a cholecystitis. There has never been a history of gall-bladder colic or biliary blockade. The history is rather that of an inflamed and infected gall-bladder in which the infection is of a mild type but capable at times of exacerbations, one of which occurred in July, 1912, and another at the onset of cold weather, and a third in February while in the hospital, as has already been stated, when she developed a slight temperature and an increase in the leukocytes. With the fall of the temperature there was a fall of the leukocytosis. The absence of nausea and vomiting is a symptom common to duodenal ulcer, cholecystitis, and pericholecystitis, though vomiting is, of course, frequent in the two latter conditions. It, therefore, gives us no help in the diagnosis. The epigastric pain, which she describes as dull and raw, is not characteristic of peptic ulcer or duodenal ulcer. It is rather more apt to occur in pericholecystitis cases.

Another very important symptom in the case is the glycosuria. We are beginning to learn, with an increased operative experience, that most cases of glycosuria after the age of forty are due to gross disease of the pancreas. Actual statistics show that 97 per cent. of such cases of glycosuria are due to pancreatic disease. Now there is a close relationship between the pancreas and liver or rather between infections of the liver primarily and infections of the pancreas secondarily.

Some years ago Dr. Ohlmacher, then Professor of Pathology at Northwestern University Medical School, noted the presence of cirrhotic pancreas in every case of hepatic cirrhosis which came to postmortem examination. He was early convinced that the same etiologic factor was the cause of the development of the interstitial processes in both the liver and the pancreas. Some operators have also discovered that after drainage of the gall-bladder mild cases of glycosuria have cleared up. We believe that in this case the diagnosis of cholecystitis is very much supported by the presence of the glycosuria. We are of the opinion, therefore, that this gall-bladder should be opened and drained and that the drainage is going to reduce materially, if not absolutely cure, the diabetes. We do not believe it to be utopian to hope that long-continued drainage will cure the case. We admit that the amount of sugar which she is showing, very nearly 7 per cent., is high and possibly nearly hopeless, but there is nothing to be gained by long-continued diet or any other form of treatment for diabetes. The patient is Jewish, and the odds are the stronger against us, perhaps, on that account. Nevertheless, I have strongly advised the patient, who came into my service, of the need for this operation and the possibilities for cure which it carries with it, and Dr. Murphy, who very kindly saw her with me, has concurred in the diagnosis and in the suggestion that the gall-bladder be opened and drained.

Dr. Murphy: We had the opportunity, two or three weeks ago, to show some of these cases where the patient had been relieved completely by drainage. One of these patients, a doctor, gained 34 pounds in sixty-seven days with simple drainage of the gall-bladder. He had an arthritis, with signs of metastases. They all disappeared with the drainage. Cultures of the gall-bladder contents were not successful.

We will expose the gall-bladder by opening the abdomen through the rectus muscle. She has a white gall-bladder, which is very common. This condition of the gall-bladder is a most important sign—a white gall-bladder with fixed adhesions, showing that she has not only had a cholecystitis, but a pericholecystitis. It is an organic adhesion following an infection.

Now we will bring the pyloric zone of the stomach and the duodenum into the field. There is no adhesion on the duodenum. Looking at the anterior surface of the duodenum, it appears to be normal. There is no evidence of ulceration. The next step is to examine the posterior surface of the duodenum. There is a nodule in the head of the pancreas in the region of the duct. That nodule extends back for fully one inch. This shows that she had a chronic infection of the gall-bladder. All that is pathologic. See the infiltration! a typical illustration of a chronic infection. That probably accounts for the lesion in the pancreas. It is a most beautiful demonstration of a pathologic condition. The pancreas is small, but woody hard. It is an infection of the pancreas which occurred from the infection of the gall-bladder. I want to feel the under surface of the pancreas to see that there is no stone in the pancreatic duct. No stone is palpable in the duct.

Now, then, we will examine the gall-bladder and see if there are stones palpable in the gall-bladder. After freeing the adhesions the gall-bladder is compressible. There are no stones in it, nor in the cystic or common ducts. We will drain the gall-bladder. The drainage will have to be kept up for a long time.

This is fine. There is the reward. We find something pathologic—not only the gall-bladder, which we recognize, but an infection of the pancreas which we believe is secondary to the infection in the biliary passages. We believe it is not a direct infection. We believe that this condition in the biliary passages is typical, namely, as long as the outlet or inlet is free you have no trouble, but immediately an obstruction occurs trouble follows.

I was very much pleased with what Dr. Mix said about glycosuria, namely, that in a large percentage of the cases it is not a nervous system condition, but that it is of pancreatic origin—that it is a definite lesion involving the pancreas. I believe we are going to come to the surgical treatment of many cases of glycosuria. This drain in the gall-bladder will be kept in for weeks or possibly even months. We will aspirate the gall-bladder. It contains a thick bile, somewhat cloudy. Then we insert the two rows of puckering-string sutures in the dome of the gall-bladder, about one-half inch apart. The rubber drainage-tube is then passed into the gall-bladder and the upper suture is drawn taut. The tube is now pushed down into the gall-bladder, thus inverting it, and the lower suture is drawn taut. Now I am going to secure the tube in position with one stitch, the needle going clear through the tube, from side to side. That is made a part of the outer row of stitches. That makes it absolutely secure. The small needle puncture immediately closes itself. The gall-bladder is dropped back into the peritoneal cavity, and the tube is secured to the abdominal wound with a silkworm-gut stitch.

This is a splendid case because it shows a definite lesion. We think we are going to get results from the drainage. We will also put in a small peritoneal drain.

Let the record show that there was no evidence of ulcer of the stomach, no evidence of ulcer of the duodenum; that the source of the cholecystic infection is not definite; that she had a typical white gall-bladder, indicative of a chronic infection; that she had adhesions between the dome of the gall-bladder and the large intestine, but no adhesions to the duodenum. The abdomen is closed in the usual manner.

Dr. Mix: March 28, 1913: The following supplementary report is made in regard to this case. She was operated on February 13th. A drainage-tube was inserted into the gall-bladder and the drainage has been as follows: February sixteenth, 120 c.c.; seventeenth, 310 c.c.; eighteenth, 100 c.c.; nineteenth, 90 c.c.; twentieth, 110 c.c.; twenty-first, 120 c.c.; twenty-second, 100 c.c.; twenty-third, 400 c.c.; twenty-fourth, 180 c.c.; twenty-fifth, 100 c.c.; twenty-sixth, 90 c.c.; twenty-seventh, 110 c.c.; twenty-eighth, 140 c.c.; March first, 120 c.c.; second, 120 c.c.; fourth, 360 c.c.; fifth, 200 c.c.; sixth, 180 c.c.; seventh, 200 c.c.; ninth, 90 c.c.; fifteenth, 90 c.c.; sixteenth, 120 c.c.

On the sixteenth the large drainage-tube was removed and a smaller one was inserted. The drainage thereafter was as follows: March seventeenth, 150 c.c.; eighteenth, 300 c.c.; nineteenth, 180 c.c.

Thereafter the drainage was no longer into a jar, but into the dressings. On the twenty-first the dressings were reported saturated. Since that time the drainage has been diminishing very rapidly. The smaller tube inserted on the sixteenth was removed on the twenty-sixth, since which time the dressings are practically dry, though there is a tiny bit of oozing which will probably cease within a day or two.

Our hopes in regard to the glycosuria have been reasonably well realized. The day after the operation, February 14th, the specific gravity of the urine was 1045; on the fifteenth, 1040; on the twentieth, 1040. A quantitative sugar analysis on the fifteenth gave the same percentage, 6.9, as had been observed before the operation. By the twenty-sixth, the thirteenth day after the operation, the specific gravity had fallen to 1025. On the twenty-eighth it was 1030; on March 2d it was 1030, and on this occasion, for the first time, sugar was absent from the urine. On the fourth it reappeared, the specific gravity being 1026 and the amount of sugar 0.4 per cent. On the fifth the specific gravity was 1018, and the sugar 0.6 per cent. On the seventh the specific gravity was 1031 in a single specimen and the percentage of sugar 1.2. On the tenth the specific gravity was 1025 and the percentage of sugar less than 0.5 per cent.

On the twelfth the specific gravity was 1021. Sugar was present, but no quantitative analysis was made. On the seventeenth the specific gravity was 1021 and sugar 1.4 per cent. On the eighteenth the specific gravity was 1020. Sugar was present, but no quantitative analysis was made. On the twenty-first the specific gravity was 1018 and the quantity of sugar 2.4 per cent. On the twenty-seventh the specific gravity was 1024, and the percentage of sugar less than 1. On March 9th the specific gravity was 1013 and no sugar was present. On March 31st the specific gravity was 1015, and it took 25 minims of urine to show a trace of sugar with Fehling's solution.

During the time which has elapsed since the operation we have not had the patient on a diet which has been very much restricted. The only thing about which we have been particular is the elimination of cane-sugar and fruit-sugar from the diet. She has, however, been permitted to have what starches she has wished. For example, at the noon meal today her diet included two soda crackers and a slice of bread. Notwithstanding this fact, she has assimilated the starches in very good shape. The abdominal pains have entirely disappeared, and she feels quite well. It is our intention to allow her to leave the hospital on Wednesday, April 2d.

CLINIC HELD BY DR. MURPHY AT MERCY HOSPITAL FOR THE CHICAGO SURGICAL SOCIETY, MARCH 1, 1913

[A demonstration of patients in the hospital and a discussion of cases previously operated, numbering, all told, 50. Many of the cases have already been published in the CLINICS, and from among the unpublished cases the following are selected for full presentation.]

DR. MURPHY: It is not my intention to do any operative work this morning. I will merely show you some of the patients operated on recently who are still in the hospital, and review some of the cases operated previously, so as to give you an idea of the work we are doing here.

CASE I.—TENOPLASTY FOR PARALYSIS FOLLOWING AN ANTERIOR POLIOMYELITIS

This patient had poliomyelitis sixteen years ago, when she was four years old. We cannot get a definite history of the original trouble, so that we were obliged to base our diagnosis on the rather meager history and the present condition of the patient. It appears that the girl was perfectly well in the afternoon, but that night she developed a fever and the following day she became delirious. When she regained consciousness two days later she had an almost complete paralysis of the right leg. When she came to the hospital the foot was inverted, and she was unable to evert it. She had power in the tendo Achillis, and to a slight degree in the tibialis anticus, showing that some of the nerve-fibers in the external and internal popliteal nerves were functionating. The peroneal group and the quadriceps group of muscles were completely paralyzed. All the extensors of the toes were active, particularly the extensor longus hallucis, which had taken on part of the function of the tibialis anticus.

We have had a number of these cases come to us in the past few years, and, therefore, we have been obliged to do some real orthopedic surgery in order to give these patients useful limbs. We are not putting mechanical supports on any of them after the tendons have become contracted and the limbs in a fixed position. This patient's foot was in extreme extension, and she walked on the outer edge of it—a talipes equinovalgus. She had no power to flex it, because the tibialis anticus was paralyzed. You can see what a splendid foot she has now. It strikes the ground flat and carries her weight well, and in place of turning in, it is turning out—that is, there is abduction instead of adduction.

We have operated on her twice. At the first operation we split the tendo Achillis longitudinally for a distance of about two inches, and divided the outer portion, consisting of about one-third of the thickness of the tendon, transversely. The inner two-thirds was divided lower down. The foot was then flexed forcibly, and the cut ends of the tendon were carefully approximated and sutured with a rather heavy chromicized catgut. The subdermal tissues were sutured over the tendon with plain catgut and the skin with horsehair. That put the entire traction on the outer half of the tendon and on the outer half of the tuberosity of the os calcis instead of on the inner half, and allowed the heel to come down.

Next, the extensor longus hallucis was exposed at the ankle through a small incision and likewise over the head of the first metatarsal bone. It was freed and divided close to its attachment to the metatarsal bone. The distal end of the tendon was sutured to the side of the extensor brevis pollicis. The proximal end was drawn out through the ankle incision, passed beneath the skin, and attached to the fibrous tissue at the base of the fifth metatarsal bone, to bring the foot into eversion. The tendons were sutured with catgut. The skin wounds were close with horsehair only.

Next the tibialis anticus was exposed and split into two parts longitudinally; one segment was divided transversely. The proximal end of this portion of the tendon was sutured into the

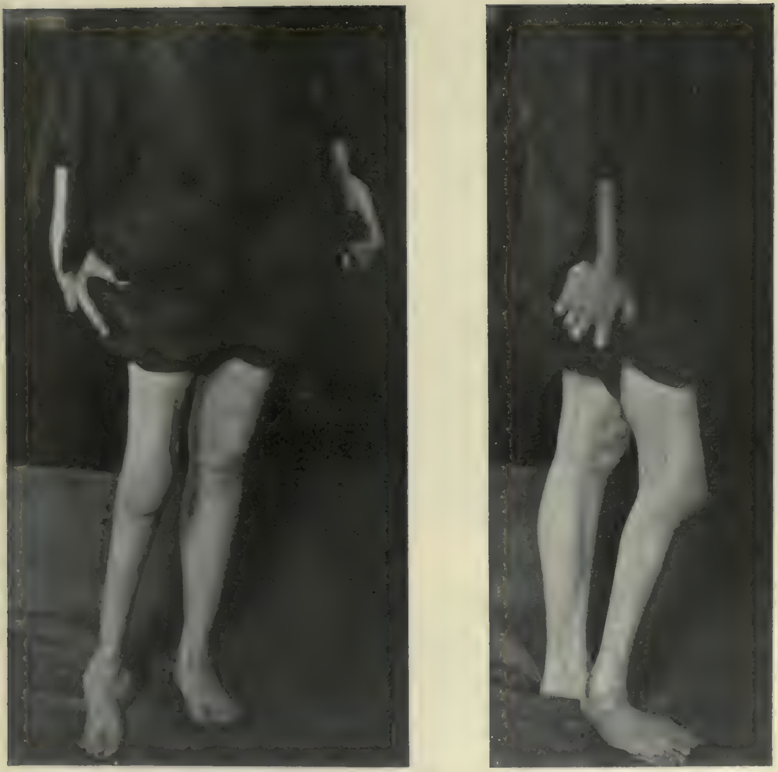


Fig. 104. Fig. 105.

Figs. 104 and 105.—Case I: Tenoplasty for paralysis following an anterior poliomyelitis. Fig. 104, position of foot before operation. The leg is very much smaller than its fellow; the foot is turned outward; the knee is below the fellow knee, because it is flexed, as can be seen in the lateral view (Fig. 105).

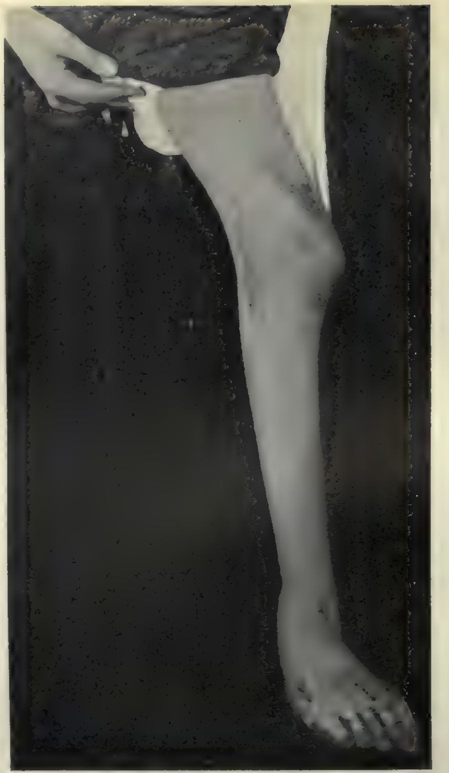


Fig. 106.

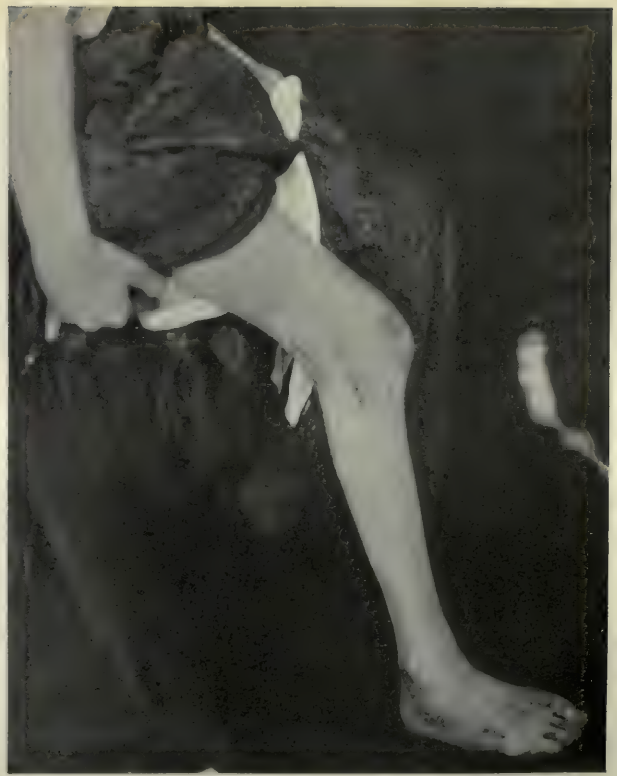


Fig. 107.

Figs. 106 and 107.—Tenoplasty for paralysis following an anterior poliomyelitis. Position of leg after operation. Foot is placed squarely on the ground and there is no inversion.

transplanted tendon of the extensor hallucis, so as to get the double effect of both muscles as everters and flexors. Then, with the tenotome, we divided the plantar fascia subcutaneously. The tibiocalcaneus ligament was exposed by an incision over the internal malleolus and shortened by reefing it. With the aid of a Jones wrench, which should be in every operating-room, the foot was manipulated until it was as flat as we wanted it. A light plaster cast was applied with the foot flat and at right angles.

The patient made an uneventful recovery, and before she left the hospital she was fitted with a shoe which would hold the foot in the proper position.

There remained a complete paralysis of the quadriceps muscle group and of the sartorius, which is supplied from the same motor center in the cord as are the quadriceps muscles—through the anterior crural. The biceps was intact. A second operation was performed later, with the idea of giving the patient a useful knee. She had no power of bringing the leg into an extended position. The biceps, although functionating, was not well developed, but sufficiently so to be used for the purpose to which we intended to put it. The biceps tendon was freed from its attachment to the fibula, drawn back through the fatty structures, and transplanted into the quadriceps tendon. In that way the biceps would be used as an extensor in place of a flexor. She is learning to use this muscle very well. If I had taken one of the muscles of the internal hamstring group, we would have a much greater distance to span. We dressed the leg in extreme flexion. The result was a very satisfactory one. She is able to use the leg and foot very well now, and as time goes on she will continue to improve. We have had equally good results in our other tenoplasty cases.

Case II.—Bilateral Tenoplasty on Feet

This is another case of the same class. This woman had a double drop-foot. We did a subcutaneous tenotomy on both the tendo Achillis, and divided and transplanted the tibialis anticus tendon in the same manner as we did in the previous case. This patient has splendid extension power in her feet, but very feeble flexion power. She did not have any paralysis of the quadriceps group.

We also divided the flexor longus hallucis. You will notice that the feet are well turned out and in good position. The operation was done only two weeks ago, so that sufficient time has not elapsed to show the final result in this case.

There is a very good field for tendon transplantation, and it is really astonishing how much one can do in the way of restoring lost muscle function by merely transplanting one or more tendons, even from an opposing muscle group. However, great judgment must be exercised if one is to be successful in this work, because, otherwise, the wrong tendon may be transplanted or the right tendon may be transplanted to the wrong place. The results obtained in this work are exceedingly gratifying. We have a considerable number of these cases, and while we have not always been able to restore all the lost function, we have succeeded in giving these patients useful limbs.

CASE III.— UNUNITED FRACTURE OF THE TIBIA AND FIBULA: TRANSPLANTATION OF BONE

This little boy, who is now five years of age, had a congenital fracture of both bones of the leg. It was some little time before the fracture was noticed, and when he began to walk there was a considerable deformity of the leg. There was a sharp angulation forward of both bones. The skiagram showed that there was some effort toward the establishment of union. There was considerable callus-formation on the ends of the fragments, but there was no bony union. He was not a very strong boy; his vitality was below par. He had been operated on unsuccessfully twice before he came to us.

We operated on him twice. The first time we did a bone transplantation, taking two fragments from the opposite tibia, and implanting one fragment into the medullary canal of the fragments of the tibia, and the other was contacted laterally with the fibula and splinted to it securely. It is not necessary to place this bone transplant into the medullary canal, but there

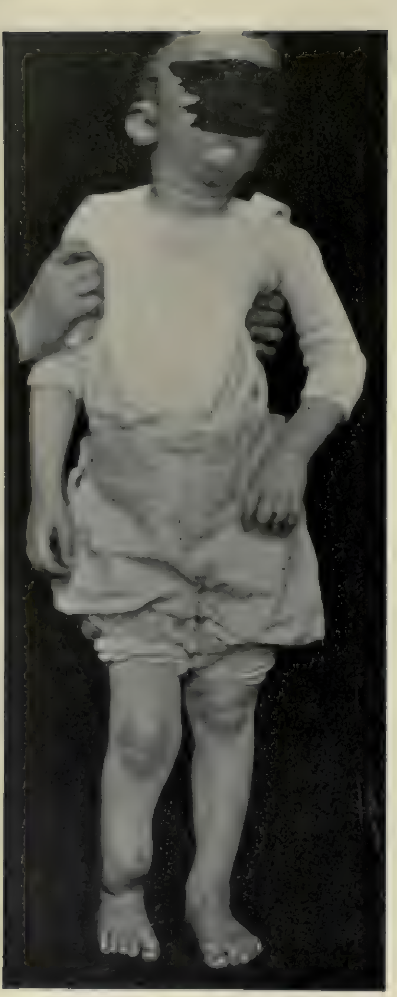


Fig. 108.

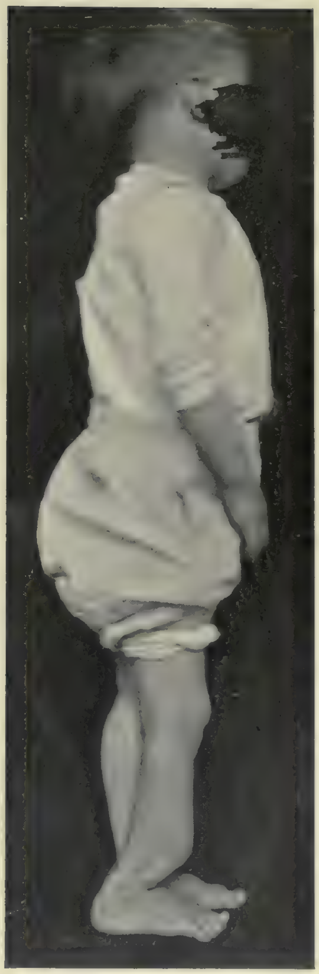


Fig. 109.

Figs. 108 and 109.—Case III: Ununited fracture of the tibia and fibula: transplantation of bone. Fig. 108, position of leg before operation. There is a marked angulation forward in the lower third, and the leg is shorter than its fellow. This can be seen by comparing the position of both knees.

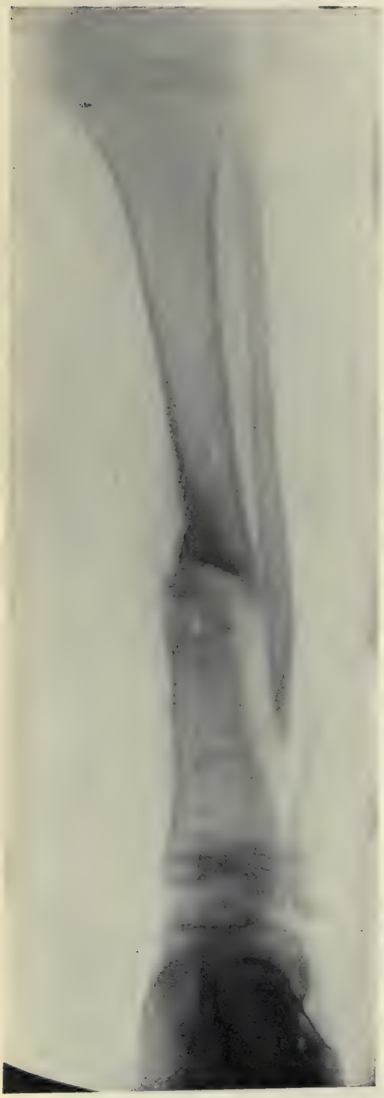


Fig. 110.—Anteroposterior view. There is apparently a bony union of the tibia, but not of the fibula. There is a separation of the fibular fragments and the upper end of the fibula is lower than normal.

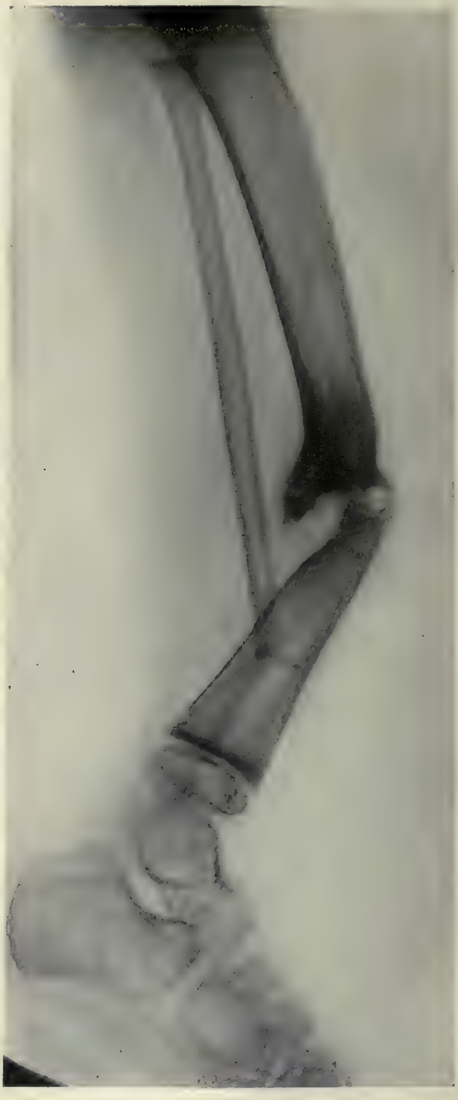


Fig. 111.—Fracture of tibia and fibula, with considerable deformity of leg. There is some effort at regeneration on the lower end of the upper fragment.

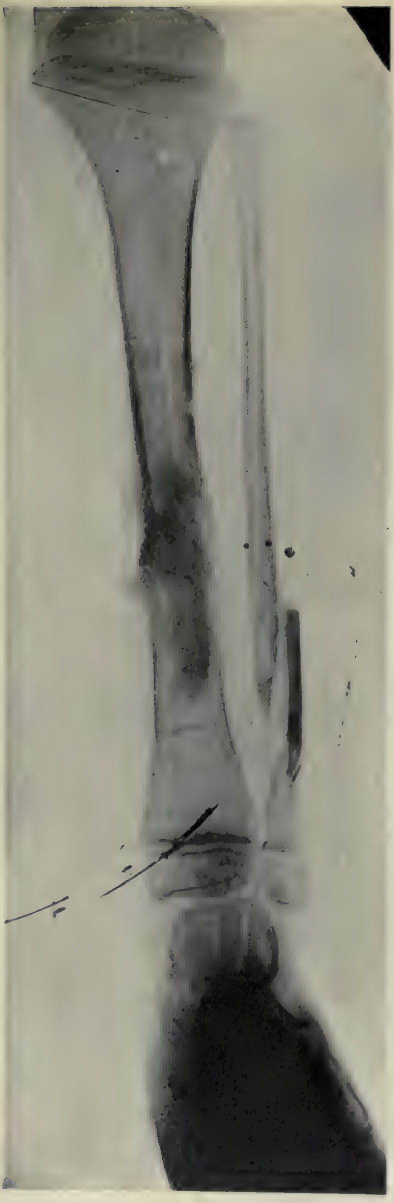


Fig. 112.—Anterior view, taken about two months after operation. The alinement of the bones is perfect. The small fragment which was splinted to the fibular fragment is not only in position, but a bony union is taking place with both the fibular fragments. The new-formed bone is seen on the inner (left) side of the transplant. The transplant in the tibia also shows up very distinctly. Regeneration of bone is also taking place here.

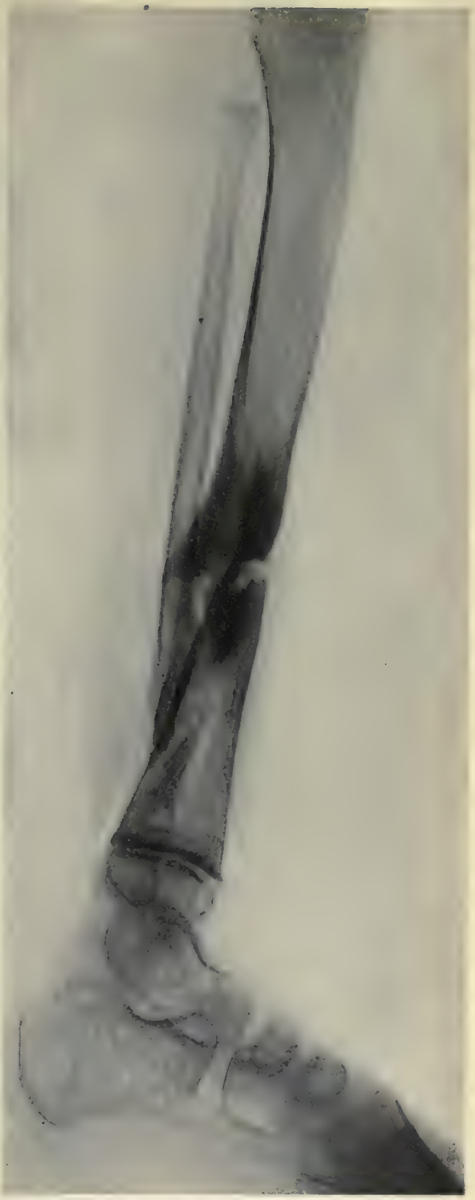


Fig. 113.—Lateral view, showing fracture of the transplant in the tibia, but no deformity.

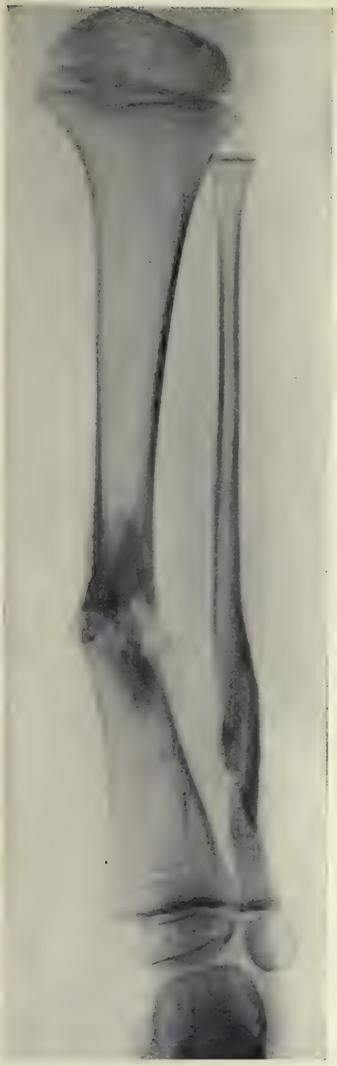


Fig. 114.—Skiagram taken six months after operation. Anterior view. There is a firm bony union of the fibular fragment. The outline of the transplant can be distinguished in the bone mass. The tibia is not in alinement, and the shadow of the transplant, which is still visible, has an outward curve to it. There is no fracture to be seen in this picture.

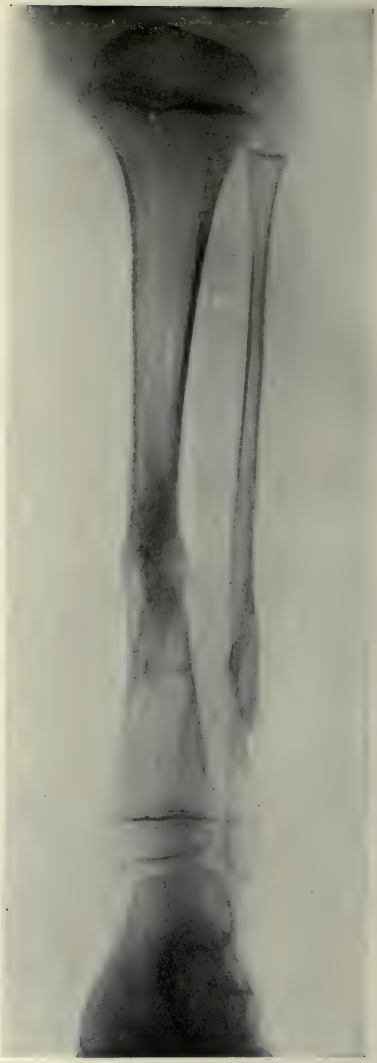


Fig. 115.—Anteroposterior view, taken four months after second operation. There is now a firm bony union between the fragments of the tibia. The transplant is still visible in the tibia. The fibula has reunited solidly. The bones are in perfect alinement.

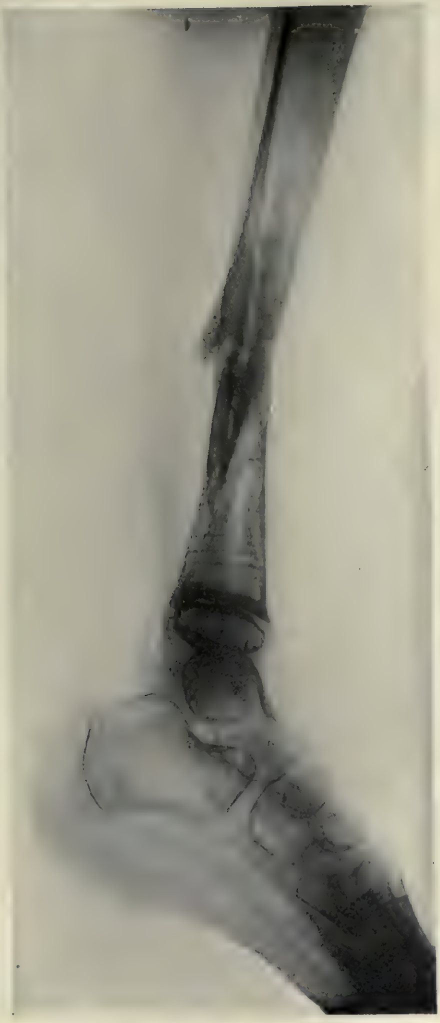


Fig. 116.—Lateral view. Confirms the presence of bony union in the tibia. Bone is in perfect alinement from every direction. The shadow of the fibula can be distinguished in this picture through the tibia. Mass of bone projecting backward (to the left) is on the lower end of the tibia.

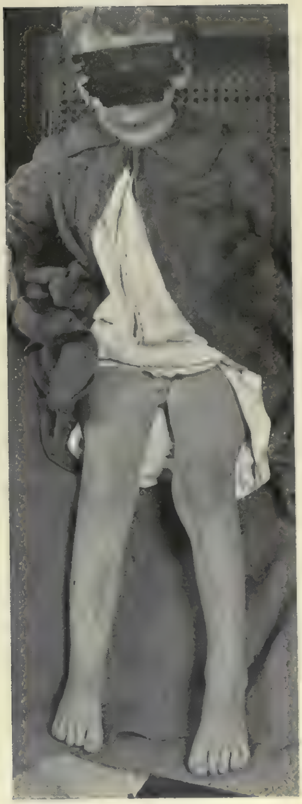


Fig. 117.—Photograph of boy four months after second operation. Leg (right) is straight and foot is placed squarely on the ground. This leg is even straighter than the normal leg.

must be an absolutely firm contacting of the transplant with living bone. If that is secured and maintained, regeneration always takes place.

The case progressed very favorably, and when the boy left the hospital, about five months after the operation, there was a firm bony union and regeneration of the new bone was progressing very satisfactorily—so much so that we removed the plaster cast which he had worn up to that time. This was a mistake, because soon after the boy reached home his mother noticed that the leg was not so firm as it was before. She brought him back to us about three months after he had left the hospital the first time, and on making a skiagram, we found that he had sustained a fracture of the transplant at the site of the former fracture of the tibia.

A second operation was performed. Another fragment was removed from the opposite tibia, but instead of placing it in the medullary canal of the fractured tibia, as we did at the first operation, we grooved the tibia and laid the fragment into the groove. The first transplant was not removed; the second transplant spans the gap between the fragments, and being in contact with living bone above and below, without the interposition of periosteum, we were confident that bony union would take place. We were not disappointed in our expectations. The boy has been in the hospital for five or six months now, and it is only very recently that the plaster cast was removed from the leg. He is not allowed to use the leg in any way whatever, and he will be kept here until we are thoroughly convinced that it is safe to let him go home. The skiagram shows a splendid result. New bone is forming rapidly and he will eventually have a useful leg.

CASE IV

Another case similar to the one just cited was that of a boy, eight years old, who had sustained a fracture of the tibia and fibula when he was one year of age. He had been operated on three times unsuccessfully before we saw him. An aluminum splint had been placed in the medullary canal of the tibia at the first operation, but union did not take place. At the two succeeding operations the fragments were wired, but union again failed to take place. Then he came to us. We did a bone transplantation operation which proved entirely successful.

When Mr. Lane was in this clinic recently he emphasized the fact that the Lane plate was not to be used in cases of non-union. That is true. We must do something more than put the bones in opposition. Bone growth has to be stimulated, and that is what we succeed in getting in this work of the transplantation of bone. In this case, as in the previous case, the fibular transplant was splinted to the fragments, and, as the skiagram shows, there is a solid bony union.

All these cases need is some substance that will conduct the osteogenetic cells across the line of fracture. We use bone taken from the same patient—an autotransplant. We usually do not remove the periosteum from the fragment, not that we believe it is essential to the regeneration of bone, but it is an advantage as a limiting membrane, and until we are convinced that it is entirely unnecessary, even as a limiting membrane, we shall continue to use it.

Of course, in the case of this boy the leg is much shorter than its fellow,—about three inches and a half,—but we feel that some of this shortening will be overcome because the lower epiphyseal line has not been disturbed.

Case V.—Fracture of the Humerus

This man came in about a year ago with an ununited fracture of the humerus. We did a typical transplantation operation, driving the transplant, which was removed from the tibia, into the medullary canal of the upper fragment, contacting it with a small sequestrum, and placing it in the medullary canal of the lower fragment, one wall of which formed the sequestrum.

The case went along in the usual manner, and after several months the plaster cast was removed. Then it was found that the lower end of the transplant was not united to the lower fragment of the humerus. The man had been working in a laundry and polishing brass work, and evidently had worked

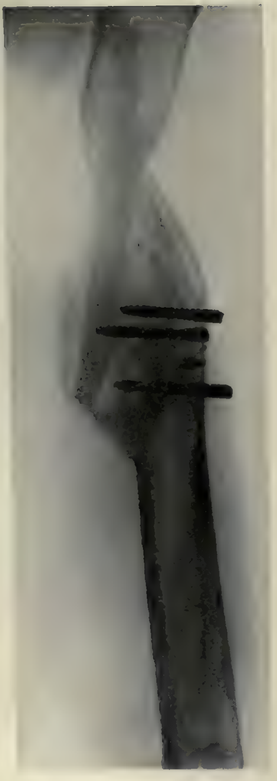


Fig. 118.—Case V: Fracture of the humerus. After second operation. Two screws hold the implant in contact with the wall of the medullary canal of the lower fragment. One screw overrides the fragment.

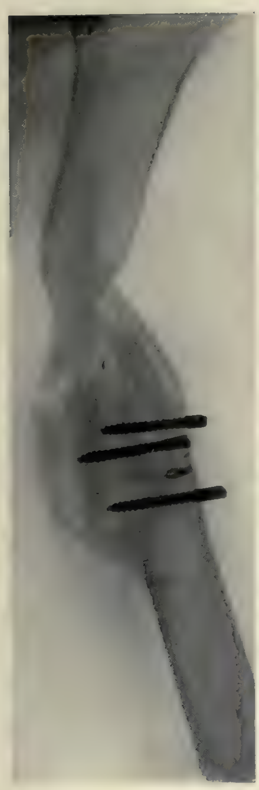


Fig. 119.—Fracture of the transplant, the result of a violent impact caused by the falling of one end of a heavy object.

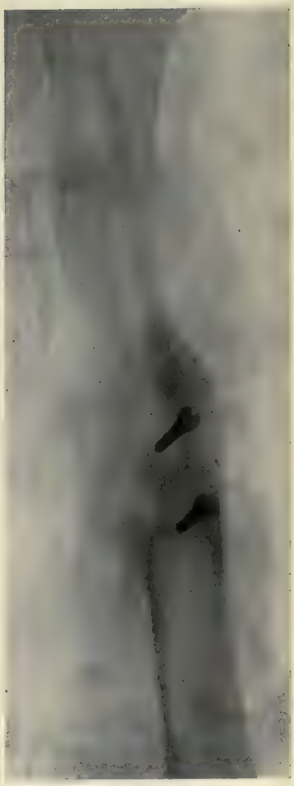


Fig. 120.—After third operation. The two fragments of the bone implanted at the first operation are seen on the left of the new transplant placed in at the last operation. This second transplant is secured to the lower fragment by means of two screws. The upper end is wedged in the groove made in the upper humerus fragment.

loose the transplant, so that it was free and union could not take place.

We operated on him a second time, and found that the transplant was firmly united to the upper fragment, but not to the lower. We did not deem it necessary to implant another piece of bone, therefore we secured the fixation of the lower end of the transplant in its bed in the medullary canal of the lower fragment of the humerus by means of three screws. Two of the screws impinged against one side of the transplant, forcing it against the wall of the medullary canal, and the remaining screw overriding it. Thus the approximation of the transplant with living osteogenetic bone and its fixation were easily secured.

About a week ago he was carrying a sofa with a fellow-workman. It was one of those big, heavy, leather sofas. The other fellow dropped his end, and when it struck the floor the entire force of the impact was given to the patient's arm and it fractured. The skiagram showed that it was the transplant that had been fractured. What to do next was the question. To ream out that compact bony tissue of the transplant was not to be considered. We decided to put in a second transplant. We removed a piece from his tibia, and it was placed into a groove that had been made on the external surface of the two humerus fragments. Fixation was secured with a screw. We are sure of a complete regeneration in this case, but we will profit by our previous experiences with this man and keep that arm in a plaster cast for many, many months. [This patient had a splendid result. The fragments became firmly embedded and united with the surface of the humerus. The skiagram shows that there is a bony union of the fractured primary transplant.—Ed.]

(For a full description of the two previous operations, see Clinics, vol. i, p. 875.)

Case VI.—Osteomyelitis of the Tibia with Complete Destruction of the Bone and an Outward Displacement of the Upper End of the Fibula

This is a case which we at one time thought was going to be a failure. We had, up to the time we saw this patient, 100 per cent. of successes with our transplant work, and for a while we announced this as the one and only failure. We were mistaken in that supposition, however, because the case is not a failure, so that we still have 100 per cent. of successes.

We have operated on this boy twice. When he first came to us we did the primary operation, which we always do in a case of this type before the bone transplantation is done. The skiagram shows that the upper end of the fibula had become dislocated outward and upward on the tibia a distance of about one inch. This deformity and shortening had to be overcome before the transplantation was made, and we had to maintain the length of the leg. We secured this result by transplantation of the upper end of the fibula into a socket prepared for it in the upper end of the tibia. We sawed off the head of the fibula just below the epiphyseal line, and transplanted it into the excavation made in the outer side of the tibia, just below the epiphysis. The fibula was secured in this position by means of a nail driven through it into the tibia. The fibula will give support to the leg and maintain its length. The epiphysis of the fibula having been removed, it leaves only the tibial epiphysis from which bone growth can take place.

We waited until union between the fibula and tibia had taken place before attempting the second operation, at which we intended to do a transplantation. When we were ready to begin the operation we were informed that the boy had an acute bronchitis. We did not operate. We waited for two weeks before we felt it would be safe to undertake the operation. At the second operation we transplanted a fragment of bone taken from the opposite tibia into the gap in this tibia. It was 18 cm. in length. It was driven into the medullary canal of the upper fragment, and contacted for a considerable part of its length with the lower fragment, which, as may be seen in the skiagram, was of such shape that it did not seem wise to do anything else than to approximate it laterally with the transplant for some of its length.

Immediately afterward the boy had an elevation of temperature, and from the fact that pus was discharging freely from the

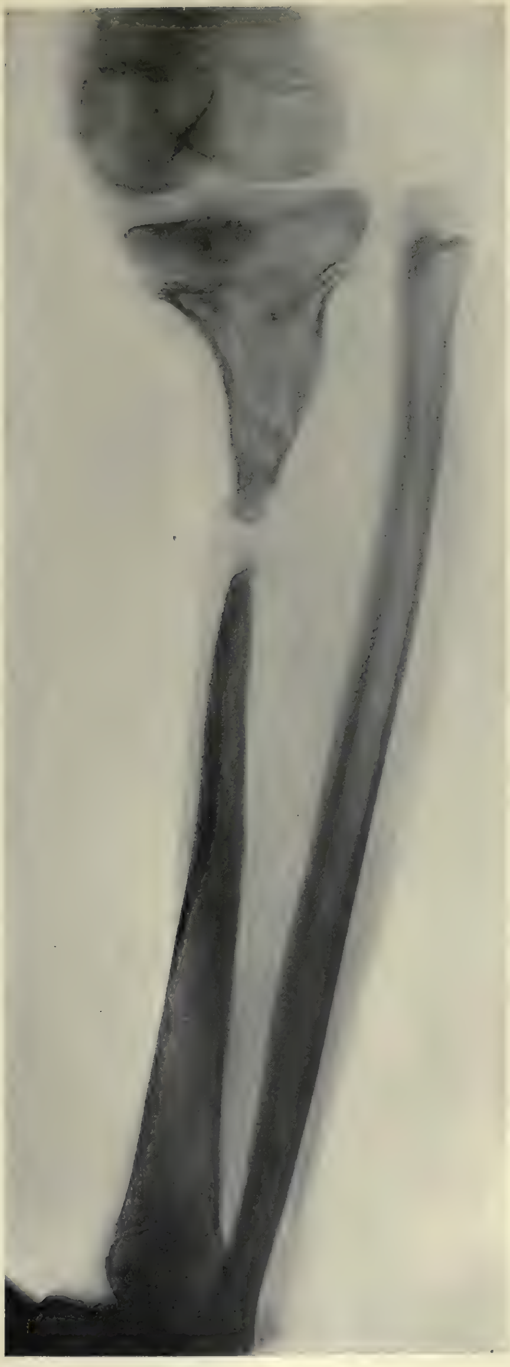


Fig. 121.—There is a separation of the tibial fragments of about one-half inch. The fibula is dislocated outward at its upper extremity, and higher than normal. There is much destruction of the shaft of the tibia.

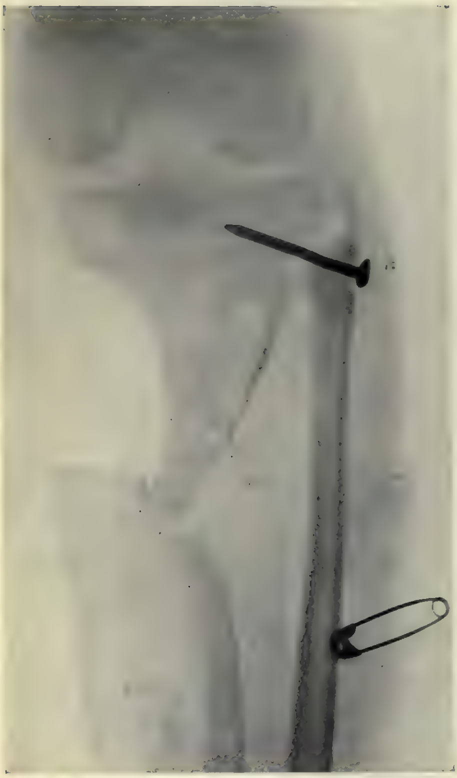


Fig. 122.—This skiagram was taken immediately after the first operation, and shows fibula nailed to the tibia just beneath the epiphysis of the latter, the epiphysis of the fibula having been removed.

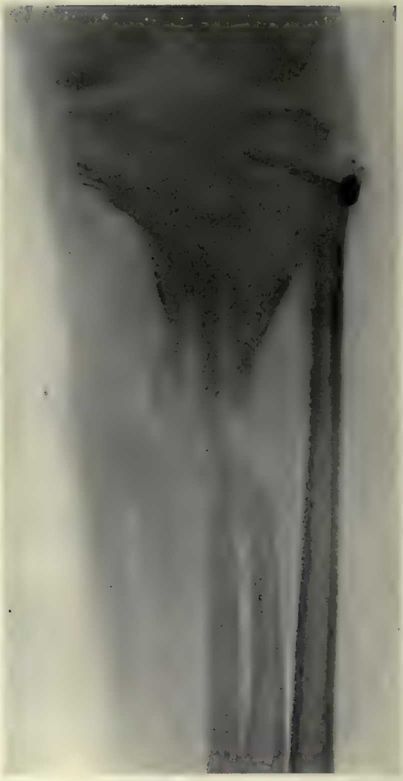


Fig. 123.—Three months after implantation of the fragment from the opposite tibia. There is some absorption of the implant at the gap between the fragments, but bone regeneration is taking place, as can be seen at the lower portion of the implant and at the lower end of the upper fragment. Fibula is firmly united to the tibia.

leg we were convinced that he had a hematogenous infection at the site of the operation, the result of his previous bronchitis, which evidently was of influenzal origin. We found the Bacillus influenza in the sputum. We felt that the bone graft would be destroyed, but by overcoming the infection we succeeded in saving the graft. We made an incision into the leg a few days ago to see whether the graft was dead, and were very much pleased to find that it is alive. It bled freely when the point of the knife entered it. Evidently the infection was not a very virulent one. The result in this case will undoubtedly be a most satisfactory one, although there is no bony union between the upper end of the transplant and the upper fragment.

CASE VII.—SARCOMA OF THE SHOULDER: REMOVAL OF THE SCAPULA

This patient was operated on about six months ago. The trouble started as a "mole" on the spine of the scapula, which some friend playfully struck. That was the only trauma that was sustained, so far as she remembers. From that time on the growth of the tumor became very evident.

My late associate, Dr. Lemke, had one of these little "moles," and when he was ill one time he traumatized it. It immediately began its malignant course. Five weeks later he was operated on, and in another five weeks he was dead, showing how very malignant these tumors are. Dr. Lemke's tumor was of the melanosarcoma type. This patient's tumor was the type of sarcoma originating in the periosteum.

We removed the entire scapula in this case, going in from in front, first taking off the long head of the biceps and the coracobrachialis, and then going in from behind and removing the scapula. We freed the attachments of the muscles very carefully and fixed them to the acromion process. This lady has very good use of her arm. She can flex the forearm and extend it with perfect ease. She can do her own hair; she can button her collar. She cannot bring the arm out on a line with the body very far, that is, abduction is limited. She plays the piano as well now as she did before the operation. There is, of course, a little deformity. The shoulder is not so wide as its fellow, but when she is dressed even that is not apparent.

There is no evidence of recurrence now, and we hope that there will be none. She is feeling perfectly well and has gained in weight.

CASE VIII.—ANKYLOSIS OF THE KNEE

This young man had a complete bony ankylosis of his knee of fifteen years' standing, the result of a metastatic arthritis followed by a purulent infection. The femur and the tibia were united the full width of the joint, so that there was nothing at all left of the joint, and the worst of it was that the leg was flexed to nearly right angles.

This ankylosis, like a majority of ankyloses, was avoidable, but in this case it was inevitable because the boy wore a plaster-of-Paris cast on his leg for two years with the knee flexed to nearly right angles. The patella had fused with the femur.

We were obliged to make an entirely new joint, restoring the normal conformation of the articular ends of the femur and tibia, chiseling through the line of bony union between the femur and the patella, and interposing fat and fascia pedicled flaps between the bones to prevent a recurrence of the ankylosis. Formerly, we made a curved transverse incision across the knee on a line with the upper surface of the tibia, but because of the occurrence of gangrene in the tip of the flap in one or two cases we abandoned that incision, and we now make two longitudinal incisions, one on each side of the patella, as was done in this case. That gives us a very good exposure of the joint, and insures perfect and early union of the flaps after operation. We have not had any flap necrosis since we are making these two incisions.

After we have exposed the joint we chisel through the bony union, remove the new formed bone, and refashion the ends of the bones to as nearly a normal contour as is possible by means of a curved chisel. The condyles of the femur, the concavities in the tuberosities of the tibia, the intercondyloid notch, ridge, and spind, are remade, so that full and complete anatomic conformation of the bone-ends is obtained in the joint; the leg is plumb,

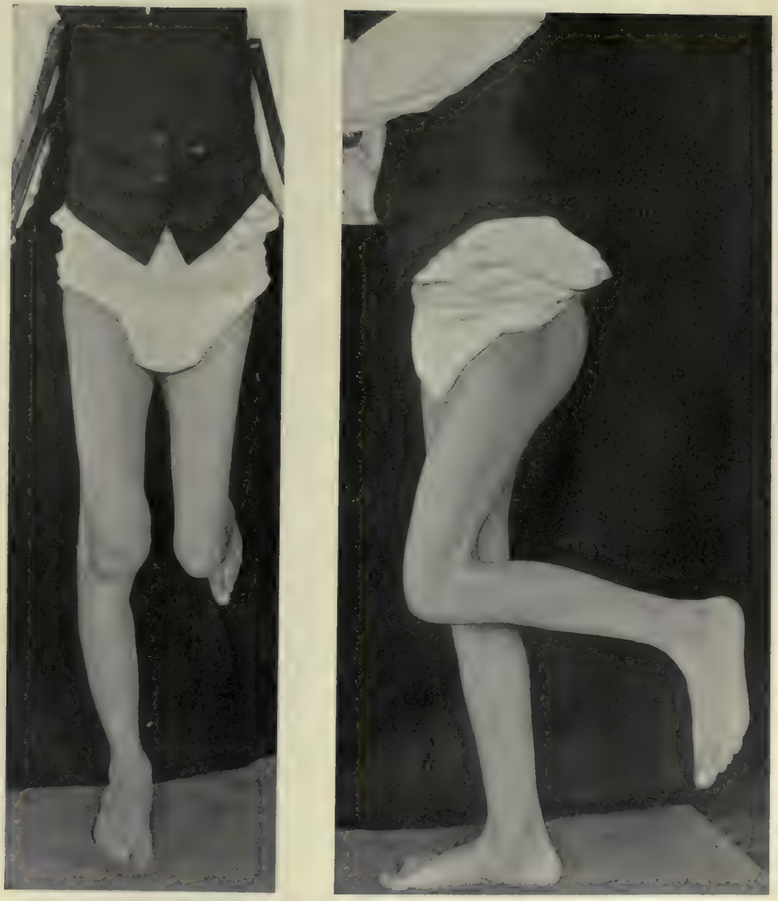


Fig. 124. Fig. 125.

Figs. 124 and 125.—Case VIII: Ankylosis of the knee. Position of the leg before operation, front and lateral views. Shows flexion to little more than right angles.

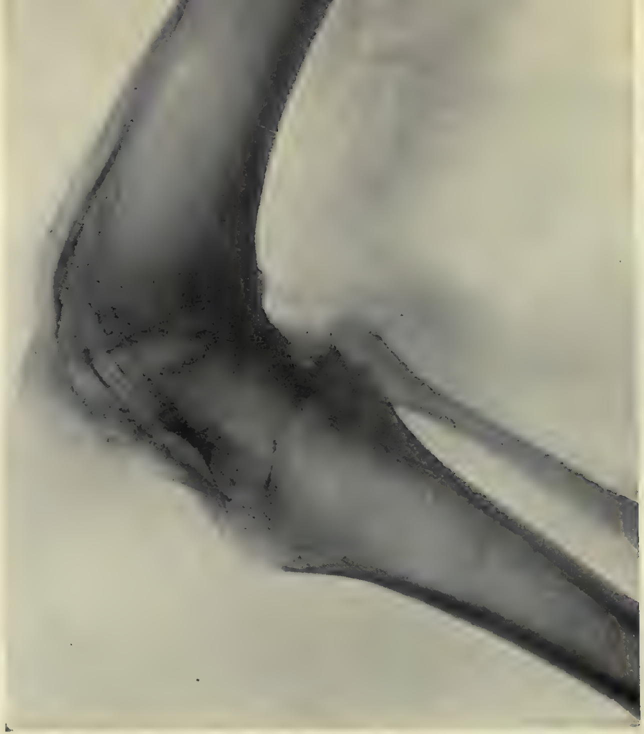


Fig. 126.—Skiagram taken just before operation, showing complete bony ankylosis between femur and tibia, the full width of the joint; likewise a bony union between patella and femur, the patella being placed low down on the femur. There is also a slight curvature of the lower end of the femur.

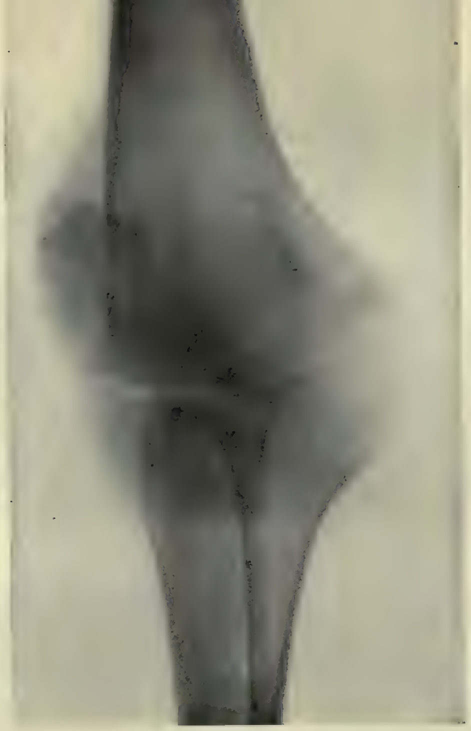


Fig. 127.—Skiagram taken about one month after operation. Leg is straight and position of bones is good. The fibula is seen to be higher than normal, because of the bone which had to be removed from upper end of tibia to make room for the interposing flaps. The bones are more prominent behind than normal, because of the position and curvature of the femur before operation.

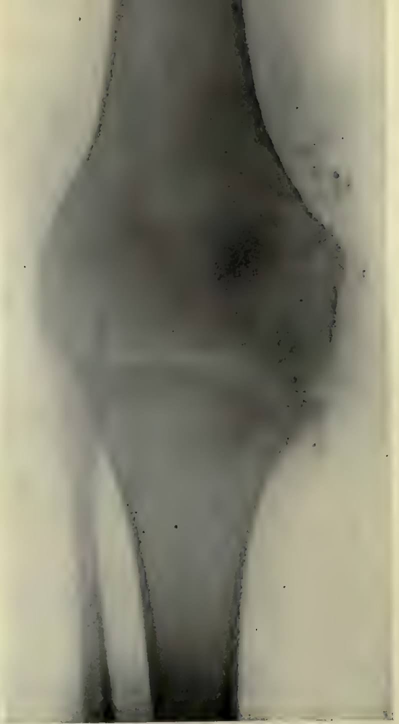


Fig. 128.—Anteroposterior view shows the new joint, the intercondyloid ridge on the tibia, and the reformation of the condyles of the femur.

and the joint possesses as much stability as it did before the ankylosis occurred. The details of the method we have described so often that you are doubtless sufficiently familiar with them to make a repetition unnecessary at this time.

The next step is to interpose the two pedicled flaps, an inner and an outer, taken from the subcutaneous fat and fascia and the remnants of the capsule. These flaps are laid across the upper surface of the tibia, so as to overlap in the midline, and are then carefully sutured in place with chromicized catgut.

The patella was freed from its attachment to the femur and was rotated on its axis 180 degrees, so that the prepatellar bursa became the lining or covering of its articular surface, entered into the formation of the new joint, and effectively prevented the recurrence of the ankylosis. All that remained to be done then was the closure of the skin incision.

It is now five weeks since this operation was performed, and that is why motion is somewhat difficult, although not especially painful. Remember, that for fifteen years he was unable to use any of the muscles which move this joint. He is now twenty years of age, so that when the trouble occurred, he had hardly fully developed his muscle action. We are, however, going to give him a good movable joint with the power of flexion beyond right angles, which is a wonderful improvement on his former condition.

CASE IX.—MULTIPLE ARTHRITIS

This patient is the wife of a physician. She has suffered from rheumatoid arthritis for ten years. She was disabled completely; nearly all the joints were involved—the knees, ankles, elbows, wrists, and the small joints of the fingers. She suffered great pain, and the joints had become deformed, so that she was unable to do anything at all. She is going home now, feeling well and free from pain or discomfort and with useful joints.

This case is a very good illustration of what can be accomplished by proper treatment. The only treatment we gave this lady was to inject into the joints a 2 per cent. solution of formalin in glycerin. This solution is not used until it is at least twenty-four hours old, although it may be older than that. It should be shaken often, so that when it is used the formalin and glycerin have mixed thoroughly. The injection is always made under gas anesthesia. The amount of the solution injected varies with the joint involved. In the case of the knee we inject 15 c.c.; the elbow, wrist, and ankle, 4 c.c.; and the smaller joints, 1 c.c. Immediately after the injection is made the joint is manipulated freely, so that the fluid is disseminated, and then a Buck's extension is applied with a weight attached. In the case of the knee we usually put on about 15 pounds, and considerably less in the case of the smaller joints; however, just how much weight should be applied will depend on the muscular development of the patient, because the weight is put on for the purpose of overcoming the contraction of the muscles, so as to keep the joint surfaces separated as far as possible.

These injections may be repeated once a week as often as is necessary. After the first injection, when the needle enters the joint, a negative pressure is made on the syringe. This shows whether the needle is in the joint cavity, and also the degree of the reaction which has followed the previous injection. If the needle is in the joint, a serosanguineous or purulent fluid will rise on the glycerin. We always use a screw syringe for making these injections, because it is very difficult to force glycerin through a syringe needle even when it is as large as the one we use—about the size of a knitting-needle.

The result from these injections is seen in this case. The patient has freely movable joints, with no pain or discomfort, and she is able to help herself as well as she ever did before this trouble began. We have had a large number of these cases, and the results have always been exceedingly satisfactory, providing there has been no bony ankylosis preceding the application for treatment.

Case X.—Trifacial Neuralgia: Division of the Nerves

This man has for a number of years suffered from severe trifacial neuralgia. He has been operated on before. He told us, when he came to the hospital, that an attempt was made to remove the ganglion, but the operation was not concluded. He did not know what happened. I presume it was severe hemorrhage; however, it made the operation which we performed a really difficult one.

These cases can be treated in a variety of ways: First, there is the injection method; second, the division method with the injection; these are extracranial procedures; third, the intracranial types of operation, the division of the roots inside the foramina, and the closing of the foramina with heterogeneous material, such as the rubber tissue of Abbe, the screws of Mayo, the ligatures, the wax plug of Murphy. Next is the removal of the Gasserian ganglion by the Hartley-Krause method,—and finally the removal of the sensory root of the ganglion—Frazier's method.

Osmic-acid injection is the plan we usually resort to first, with the division of the nerve at the intracranial foramina. It gives a relief varying from fourteen to eighteen months. Patients who have gone as long as seven years without recurrence are considered cured. A number of them have gone three years, a smaller number four years, but the average case recurs in from fourteen to eighteen months. The second osmic-acid injection gives about the same period of relief as the first. The third and fourth injections are not so successful. The alcohol injection gives relief for from six to ten months. Whether the injection is given above the zygomatic arch or below, it seems to make but little difference. To us the injection below the zygomatic arch seems a safer and more certain procedure.

We have done altogether some 16 or 18 Gasserian-ganglion removals, that is, complete removals, securing in all cases in which we have been able to get reports permanent relief. It is the desirable operation if it can be executed, but we have found it difficult to execute on account of the bleeding, and therefore we have resorted to the easier and safer procedure of intracranial extradural exposure of the inferior and superior maxillary division of the nerve. We have divided them and placed a paraffin or wax plug a little larger and thicker than a nickel over the foramina, and pressed it in and firmly interposed it between the divided ends of the nerve in that position. The pivots which are made by pressure of the paraffin prevent the material from sliding, and those pivots are in the foramen ovale and foramen rotundum. The superior maxillary division is not disturbed in this procedure.

In the removal of the Gasserian ganglion the roots are not divided until the upper layer of the dura is entirely freed from the ganglion; then the roots are divided and the Gasserian ganglion evulsed, holding both of the roots in the same root-retracting forceps, which is of the pattern of the Mayo cystic or common-duct forceps. It makes a splendid evulsor, for the depressed root is removed when it passes through the upper half of the dura on the surface of the ganglion, and then backward and upward over the temporal elevation until the posterior root is seen shining in the field. This is hooked up with a blunt hook resembling that used by Alexander in extracting the round ligament, or by the oculists in doing myotomies on the eye-muscles. When the root is thoroughly hooked, traction is made on it. It is not cut off—just simply dropped, because it has been detached from its central position; it never reunites because there is a permanent degeneration of the part, that is, the axons of the pons, having no neurilemma, are permanently incapacitated, as was shown in the experiments of Frazier.

A starch headcap is put on these cases and not removed for two weeks.

Case XI.—Abscess of the Liver

This patient is under the care of Dr. Milton Mandel, and I have asked the doctor to discuss the medical phase of the case. He asked me to see the case with him this morning.

Dr. Mandel: The patient I am presenting entered the hospital in the medical service February 17th. She is an Italian, thirty years of age, and does not speak English, so that her history is necessarily incomplete. Four days previous to entering the hospital patient had a chill lasting twenty minutes, followed by a pain in the right axillary region, which was aggravated by coughing, breathing, and moving. She also had a cough, which at first was unproductive, and a high fever.

Physical examination made on the day of entrance revealed a lobar pneumonia involving the right lower lobe, and a slight transverse enlargement of the heart, with accentuation of the second pulmonic tone. Abdominal examination was entirely negative. Temperature, 103° F.; respirations, 40; pulse, 120; leukocyte count, 20,000.

February 19th the patient had what was apparently a crisis. Temperature, pulse, and respirations dropped to normal, and the toxemic disturbance, which had been intense, rapidly subsided. For ten days she continued to improve; temperature remained normal, strength increased, and appetite returned. She was apparently convalescing rapidly.

February 26th she again developed a cough, which on the following day was productive. The expectoration was of brick-dust color, and distinctly purulent, but absolutely odorless. Examination of the lungs at this time failed to reveal an explanation.

February 27th, at 8 P. M., the patient had a chill, and the temperature, which had been normal for ten days, was 103° F. I saw her at 10 P. M. She was greatly prostrated, temperature was high, pulse 140 and thready, respirations very rapid and shallow. With the exception of distant and somewhat high-pitched breath-tones over the right lower lobe posteriorly, lung findings remained negative. The liver, however, which previously had not been palpable, was now distinctly enlarged and extremely tender. This was especially marked on making pressure upward on the lower ribs from behind. These abdominal findings were positively not present forty-eight hours previously.

Regarding diagnosis, several conditions must be taken into consideration: Inasmuch as this complication, if it is a complication, is postpneumonic, we would naturally think first of lung abscess or empyema. However, I think we can exclude both conditions by the history alone. It is well known that in postpneumonic abscess or empyema the temperature approaches or may reach normal, but in a few hours again becomes elevated, and later of a hectic type. In abscess we get the mouthful expectoration of offensive pus entirely unlike that which this patient evacuated, and usually the physical signs of cavity. Empyema would give us the findings of an effusive pleurisy. Delayed resolution, gangrene, and echinococcus need not be seriously thought of. We must, therefore, consider conditions below the diaphragm to explain the picture, and I have absolutely no hesitancy in making a diagnosis of liver abscess rupturing through the diaphragm and into a bronchus.

It is hard to conceive how she got her abscess. She has never had dysentery (amebic or bacillary), typhoid, or any other gastro-intestinal disturbance. It is not likely that the inflammatory process extended from the lung through the diaphragm into the liver. It would be possible, in a case of pneumococcus sepsis, to have a metastatic focus in the liver, but I do not believe that this has occurred, as the pneumococcus infection had apparently entirely subsided. There have been no evidences of empyema, of gall-bladder or septic cholangitis, so that extension by contiguity can be excluded.

I believe, therefore, that we are dealing with a liver abscess. Where it came from I do not know. It is barely possible that at some time in her life she had a dysentery which was not diagnosed.

The condition of the patient this morning does not permit of an extensive examination. You will note, however, that she is icteric, that the edge of the liver extends a hand's-breadth below the costal arch, and that she flinches on slight pressure or heavy percussion.

Visiting Doctor: Might it not have been a liver abscess from the beginning?

Dr. Mandel: No, sir! The physical findings were typical of pneumonia. The leukocyte count, which was above normal, dropped to 8000 after the crisis. This morning it is 30,400. Furthermore, the original diagnosis was confirmed by blood culture. This condition developed in the last forty-eight hours. The abscess may possibly be of long standing. They are sometimes quiescent for long periods.

Dr. Murphy: She came into my service today. What will

I do? That woman has just had a rupture of the abscess into the lung. This is the condition exactly. The tension of the abscess has been relieved by this rupture into the lung. If we operate now—today—she will die. We have learned from Dr. Ochsner that when they are in that acute stage they should be tided over, possibly for a week or ten days, before the abscess is opened. That is the real situation. That removes the most dangerous symptoms; then you can operate with not one-twentieth of the risk, not even one-fiftieth of the risk, that you would have if you operate today. I think the diagnosis is correct. It is a liver abscess. The surgical management must be to wait until she has regained her strength, now that the tension is relieved, before attempting any operation.

Other cases shown were the following:

CASE XII.—RHEUMATOID ARTHRITIS

Here is an example of another class of cases—a rheumatoid arthritis. This man is a farmer. He was working on his farm when he had a chill, after which he developed rheumatoid arthritis. He was badly crippled. Previous treatment was ineffective. His knees were so badly crippled he could just hobble around on crutches. We gave him injections of formalin and glycerin and of mixed vaccines. We did not know what type of infection he had had, so that we could not make an autogenous vaccine, which we always do when we know the type of the infection and can make a culture. We gave him the mixed vaccines, and the reaction was so great that we thought he was going to die, showing that the vaccine was just what he needed. If you do not get a reaction following the injection of any vaccine, you are not in the zone of the type of bacteria which produced the infection. The reaction must be a definite one. This man has had only one injection of formalin in glycerin in each knee, and following the injection we put on a Buck's extension. What I want to call attention to is the fact that the vaccines nearly killed him. He had a temperature after the first injection of something like 105° F. He improved rapidly under the vaccine treatment, and he is now able to use all his joints with perfect freedom of motion. He is free from pain or discomfort, showing what vaccines will do. We use them very extensively in our work. We prefer the autogenous vaccines and always use them when we can.

You can give tuberculin to a non-tuberculous patient in doses of 1000 minims of a 10 per cent. dilution and get no result, but give a large dose to a tuberculous patient, and see what happens: the patient will almost die from the reaction. I recall a case where a doctor wrote me about what dose of tuberculin he should give a patient. I asked one of my assistants to send him the instructions, but the doctor did not wait to receive them. He went to a drug-store, bought a bottle of Koch's old tuberculin, full strength, and gave the patient the full bottle—fifteen minims. He called me up that night because the patient was unconscious, with a temperature of 106° F. She remained unconscious for five or six days, but she did not die.

(Cases XIII and XIV will be published in full later.)

CASE XV.—OSTEOMYELITIS OF THE TIBIA WITH EXTENSIVE DESTRUCTION OF THE BONE.
(See CLINICS, vol. ii, pp. 305-312.)

CASE XVI.—CHONDROSARCOMA OF UPPER END OF FEMUR.
(See CLINICS, vol. i, p. 299.)

CASE XVII.—DISLOCATION OF SHOULDER.
(See CLINICS, vol. ii, pp. 413-420.)

CASE XVIII.—SARCOMA OF UPPER END OF TIBIA.
(See CLINICS, vol. i, p. 299.)

CASE XIX.—OSTEITIS FIBROSA CYSTICA.
(See CLINICS, vol. i, p. 555.)

CASE XX.—DIVISION OF THE BRACHIAL PLEXUS WITH SUTURE: RESTORATION OF FUNCTION.
(See CLINICS, vol. ii, p. 259.)

CASE XXI.—PLASTIC OPERATION ON EAR.
(See CLINICS, vol. ii, p. 389.)

CASE XXII.—ARTHROPLASTY ON HIP.
(See CLINICS, vol. ii, p. 449.)

CASE XXIII.—FRACTURE OF ANATOMIC NECK OF FEMUR.

(See CLINICS, vol. ii, p. 421.)

CASE XXIV.—FRACTURE OF HEAD OF FEMUR.

(See CLINICS, vol. ii, p. 22.)

CASE XXV.—FRACTURE OF THE NECK OF THE HUMERUS.

(See CLINICS, vol. ii, p. 137.)

CASE XXVI.—FRACTURE AND DISLOCATION OF THE CARPAL BONES.

(See CLINICS, vol. ii, p. 393.)

CASE XXVII.—ANKYLOSIS OF JAW.

(See CLINICS, vol. i, p. 905, and vol. ii, p. 405.)

CASE XXVIII.—DISLOCATION OF SEMILUNAR CARTILAGE.

(See CLINICS, vol. ii, p. 435.)

ACUTE SUPPURATIVE PROSTATITIS

Early Drainage into Urethra; Subsequent Leakage through Capsule, with Infection of the Perirectal Tissues; Ischiorectal Abscess; Incision, Breaking Down Partitions between Pus-pockets, and Drainage; Unimpeded Recovery

HISTORY

Dr. Murphy: This case is referred to me by Dr. Edward F. Wells, of this city, who is present this morning. I will ask the doctor to take up the history of the case.

Dr. Edward F. Wells: The patient is sixty-two years of age, 5 feet 4 inches tall, and ordinarily of 215 pounds weight. Had a Neisserian infection in early life; at thirty-eight had severe nephritic colic. At fifty-three he suddenly became jaundiced, which soon became profound; there was tenderness over the gall-bladder, without pain or fever. After four months he had, January 16, 1905, a severe chill, with temperature reaching 104° F. This was accompanied and followed by a profuse, gradually lessening, flow of saliva. The pulse was 62; leukocyte count, 8800, with 63 per cent. of polynuclears; sugar was absent from the urine; the gall-bladder was greatly distended, as shown by palpation and a radiogram; following a test-meal the stomach-contents contained a normal amount of free and combined HCl, with moderately reduced motility; the systolic blood-pressure was 125; his weight was 182 pounds.

In February, 1905, he was operated on by Dr. Murphy. The gall-bladder was greatly distended and discharged an enormous quantity of bile; it was anastomosed to the transverse colon by means of the Murphy button; no calculi were found in any of the bile-ducts; but the common duct was completely obstructed as it passed through the head of the pancreas; the pancreatic caput was greatly enlarged, very hard, and was supposed to be affected by a malignant growth. Recovery from the operation was fairly rapid, and he soon regained his former weight. For several years the bowels remained uncomfortably loose, with, very gradually, a partial accommodation to the new anatomic conditions. In April, 1911, he had a severe cholecystitis, with chills, fever, leukocytosis, tenderness, prostration, slight jaundice, and rapid loss of weight. In November last, while out of the city, he had chills, fever, and epigastric distress for several days.

During the night of December 17, 1912, he was awakened by a severe chill, followed by temperature of 103° F., pains in the back, and frequent micturition. When seen the following morning, urine was voided only with difficulty and much pain; the urine contained considerable pus and many red blood-corpuscles; the prostate was notably enlarged and exquisitely tender to the palpating finger; the temperature was 102° F. The fever subsided; the frequent, painful, and difficult urination continued to be more or less distressing until January 24, 1913, when he was unable to void urine. At this time 18 ounces of urine was removed by the catheter, since which time he has passed no urine voluntarily. A few days before beginning the use of the catheter, and subsequently, the urine contained but little pus, and the rectal palpation of the prostate became more painful. Daily the bowel movements increased in distress, and since the evening before last there has been a throbbing rectal pain, with a notable swelling to the right of the rectum. There is a moderate leukocytosis, with no increase in the proportion of polynuclears, and there is no material elevation of the temperature.

COMMENTS AND OPERATION

Dr. Murphy: I wanted the whole history of this case because my recollection of it was that I thought his primary lesion in 1905 was probably a carcinoma of the pancreas and I wanted to put myself on record because I was mistaken. When we operated on him we found an enlarged gall-bladder, which we anastomosed to the transverse colon. This condition subsided as soon as the gall-bladder was drained. The obstruction of the common duct was probably due to an acute infection of the pancreas. There was no stone to be found at that time. We always drain these acute infective cases. Years ago, in our original article on this subject, we stated that these cases should be drained. Doing cholecystectomies for obstruction to the common duct were unheard of up to that time. Now we have developed gall-bladder surgery to such a degree that we remove everything. Draining the gall-bladder into the large intestine with the button is the method that Kocher advises. At that time it was feared that that procedure would cause an infection of the bile-ducts, but there is not one single recorded case where such an infection occurred. When the drainage through the ducts is free, there are multitudes of infective microörganisms that come in and go out of them. That is in consonance with our clinical observations and results.

An interesting feature in the case today is that this man had an infection in his prostate which has extended into the periprocteal tissues. You can see how this and the irritation of the bladder produced considerable quantities of pus in the urine.

The question is, did the infection extend from the prostate into the bladder, or did it remain localized between the bladder and the prostate? We are going to examine him under anesthesia. Will I find the infection leading from the prostate into the perineum and is there a sinus leading back into the intestine? These are the points which I have in mind at this time. Occasionally, an ischiorectal abscess comes through the lower part of the perineal ligament and appears in the perineum. These cases are not difficult to handle unless they are tubercular in origin. When the abscess is in the cellular tissue and the perineal ligament, it is usually extremely wicked. When the infection passes through the ligament or through the floor of the rectum into the periprocteal tissue, the operation is not nearly so difficult. Judging from the position of the abscess, I would say that it is a procteal lesion. If this was a man of thirty or thirty-five, it would not be a serious case, but in a man of sixty-two it is a serious lesion. Every infection of colon bacillus type in that portion of the body is serious.

We are going to dilate the sphincters first. The infiltration is high up—as far as I can reach. The pus is escaping from the prostate. I cannot feel any place where the rectum is fixed to the prostate. This infection is coming from the prostate. It is a urinary infection, rather than a rectal infection. That is not so bad for the patient.

There is the pus flowing out on my finger. That gives you an idea of the size of the pus-cavity. There is an infiltration of all this tissue which I dislike very much. There are a number of pockets of pus here which I can feel with my finger in the rectum. I feel convinced now that this is a prostatic infection. We will put in a rubber drainage-tube. What have his toxic symptoms been?

Dr. Wells: He had no fever until yesterday. All you see is of recent development.

Dr. Murphy: I believe this lesion started from his prostate. I think our interpretation was correct, namely, that the pus was not present in the beginning—that the pus which appeared in the urine was from a prostatic infection that was draining into the bladder. His chances are enormously better than they would be if he had a phlegmon of colon-bacillus origin. What was the leukocyte count?

Dr. Wells: The leukocyte count a month ago was 10,000.

Dr. Murphy: That is important always in cases where you have no temperature response. Many times the leukocyte count tells the story of the infection. Many times there is a leukocytosis when there is no temperature response to the infection. The drainage-tube will be fixed to the skin with a silkworm-gut stitch.

[Following the operation there was a moderate drainage of bloody serum, with but little pus. The swelling quickly subsided; after the fourth day the urine was voided naturally; the drainage-tube was removed in a week, and healing rapidly ensued; recovery complete.—Ed.]

SURGICAL CLINICS
OF
JOHN B. MURPHY
Volume II Number 4

SOME OBSERVATIONS ON VACCINE AND SERUM THERAPY FROM DR. MURPHY'S CLINIC

BY PHILIP H. KREUSCHER, M.D., OF DR. MURPHY'S STAFF*

As early as 1880 scientists demonstrated by experiments that bacteria injected into the blood-stream of healthy animals were destroyed by some then unknown specific action of the blood of the animal. In 1885 Fodor injected large quantities of pathogenic bacteria into the blood of animals, and found that the blood was sterile in forty-eight to seventy-two hours. In 1887 he demonstrated that the blood of guinea-pigs was destructive to living bacteria. It was concluded that this was due to some specific toxin which was supposed to be present in the blood of healthy animals. Metchnikoff and his co-workers after years of research advanced the theory that certain leukocytes (phagocytes) had the intrinsic action of taking up, destroying or digesting living pathogenic organisms. This they are enabled to do by the opsonins which in some way combine with the bacteria and prepare them for destruction by the phagocytes. Dr. A. E. Wright demonstrated that fact, and first used the term "opsonins." He also discovered that these opsonins may be increased by injecting bacterins or bacterial vaccines, and thus an entirely new method of treating infectious diseases was introduced to the scientific world.

Bacterial vaccines are designed to antagonize bacterial infections. This may be accomplished in a prophylactic manner, as illustrated by the antityphoid inoculation, or therapeutically, as by the use of pyocyaneus vaccine in a case of pyocyaneous infection. Every healthy animal organism antagonizes pathogenic bacteria, but when the animal of itself does not possess the *suf-*

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*Read before the South Side Branch of the Chicago Medical Society, May 27, 1913.

ficient antitoxic power, it becomes necessary to increase the so-called phagocytosis by artificial means. The factor which produces this active resistance against the destructive hosts is known as immunity. When the animal organism, by its own power, successfully destroys the infection, we have spontaneous recovery. Under ordinary circumstances every living animal is constantly exposed to possible sources of infection. Bacteria are present in the air we breathe, in our food and drink, as well as on our skin and in our alimentary canals. It is obvious, therefore, that there is some potent natural means of resisting the attack of the organisms, and that it is only when these means fail or are insufficient that infection takes place. This resisting power against the invasions of microörganisms is termed immunity—the exact opposite of susceptibility. Furthermore, the process of natural cure of any infectious disease is made possible by such a degree of immunity as may suffice to destroy the invader.

There are two varieties of immunity—the natural and the acquired. Natural immunity is that which is inherent in the constitution of the animal when born, and not due to any event taking place in its life-history. Thus the lower animal is immune to gonorrhea, and the human is immune to the germ of chicken cholera.

The causes which lower the resistance or immunity are of two kinds—general and local. Of the general causes, exposure, starvation, and malnutrition are the most important. Local causes, including injuries, contusions, or irritations due to chemic substances, lower the natural resistance.

Acquired immunity is of two kinds—active and passive. Active immunity results from a previous attack of a disease contracted naturally or due to artificial inoculation. Syphilis and the exanthemata are good illustrations of diseases conferring an active immunity, although even these are not always absolute.

Passive immunity is that which is conferred on an animal without effort on its part, namely, by the injection of serum from an animal which has already acquired an active immunity against the disease in question. If some of the serum from a horse which has been actively immunized against tetanus is injected into another animal, the latter will also become immune to the tetanus bacillus or to its toxin. Passive immunity cannot be bestowed by the injection of serum from an animal which is *naturally* immune. When the animal is immune, it contains in its make-up protective substances commonly known as antibodies, which are present in the blood or in the tissue secretions. Among them we may include antitoxins which act by neutralizing the bacterial poisons, and bacteriolysins which destroy the bacteria, and opsonins which in some way prepare the bacteria for destruction by the phagocytes. In contrast to these are the antigens, which are substances that stimulate the production of antibodies. Bacterial vaccines, living or dead, are antigens. The fusion of the diphtheria bacillus used to stimulate the production of diphtheria antitoxins is likewise an antigen. Wright and Douglas employed the term opsonins to designate the elements in the blood-serum which prepare the bacteria for destruction by the phagocytes. By counting the bacteria contained in a certain number of leukocytes and dividing to obtain an average, the so-called phagocytic index was obtained. The phagocytic index of the patient, divided by the average phagocytic index of a given number of normal persons, gives the opsonic index. The determination of this index requires time, skill, and a well-equipped laboratory, and is not considered indispensable to success in this new department of therapeutics.

Wright was first to determine the phases of reaction by means of the opsonic index. A short time (twelve to twenty-four hours) after the inoculation is made the opsonic index falls lower than it was previous to the injection. This he designated the negative phase. After an interval of two or three days the opsonic index rises above the starting-point. This he called the positive phase. After this there comes a point of sustained high tide of immunity. Thus the injections are repeated from time to time, and the positive phase gradually attains a higher level, until it may be as high or higher than that of a normal individual. In other words, if the vaccinations are properly given (never during the negative phase), the patient's tissues are stimulated to an increased production of the opsonins, phagocytosis is increased, the invading bacteria are disposed of, the patient recovers from the infection.

Theoretically, the opsonic index is a valuable guide in determining the size and frequency of the dose of the vaccine, but the process of estimating the index is so intricate, so tedious, and so liable to error in the hands of the average clinician that it is not practicable in our every-day work. It is our conviction, however, that in certain obstinate cases, in which the therapeutic vaccine injections have wholly or partially failed, the study of the opsonins may furnish a most valuable clue to the cause of failure. In practice the clinical symptoms, such as rise in temperature, increased pulse-rate, the amount of local pain, and the inflammatory reactions in the localized infection, and the patient's general condition may be relied upon in determining the size and interval of the dosage.

Our general rule has been to begin with the small dose and progressively increase, immunity being more effectually produced by the repeated injections of gradually increasing doses than by a single injection of a large dose. Should no improvement be noted, the size of the dose may be increased or the interval shortened, or both. If a pronounced clinical reaction occurs, characterized by general malaise and an aggravation of symptoms, it indicates that the dose has been too large. In all our joint cases which receive the combined treatment of vaccine injections and the formalin and glycerin injections into the joint, we have made it a rule never to inject a vaccine on the day preceding or during the three or four days following the joint injections. As a general rule, the intervals between the doses in the more acute or subacute infections vary from three to four days. After the acute symptoms have subsided, the interval may vary from four to five days. In other words, as stated above, never inject during the negative phase when the opsonic index is low, but wait for the reappearance of the high tide of the positive phase.

It is obviously impossible to discuss more fully the various complex steps of the subject of immunity. The physician who wishes to familiarize himself with work along this line may find exhaustive references in the German work of Wolff-Eisner, or Ricketts' "Infection, Immunity, and Serum-therapy." The work of Citron has recently been translated into the English language, and is very concise and practical.

Definition and Method of Preparation.—Bacterial vaccines are suspensions of definite quantities of dead pathogenic bacteria in physiologic salt solution.

Unless the best kind of culture-medium is used, the vaccine will be worthless when the subsequent steps are performed. Bacteria are so sensitive to their environment that it is very easy to attenuate a virulent strain by two or three transfers on unsuitable media. Usually the medium which gives the most prolific growth in the shortest time is the one which *attenuates the culture least.*

About 95 per cent. of all cultures for vaccines are grown on plain agar, blood-agar, or hydrocele fluid. Plain agar must be made from meat infusion (not from the commercial extracts) from which the muscle-sugar has been removed by inoculation with the Bacillus coli. To this there are added 15 gm. of agar-agar, 10 gm. of Witte's peptone, 5 gm. of sodium chlorid, 1 gm. glucose for each 1000 c.c. of the finished product. This is titrated and adjusted so that it takes 1 c.c. of 0.1 per cent. normal NaOH to neutralize 10 c.c. of the agar. This is tubed in 20 c.c. test-tubes with 10 c.c. for plating, and part put with 5 c.c. for slants and then sterilized in an autoclave.

Blood-agar is prepared by adding 1 c.c. of sterile human blood to 5 or 10 c.c. of agar which has been melted and then cooled to 50° C.

Hydrocele fluid agar is made by adding 1 part of the fluid to 2 parts of 2 per cent. agar in the same way.

We use the glucose agar when we expect to find Staphylococcus pyogenes aureus, albus, or citreus, B. coli, B. typhosus, B. proteus, or B. pyocyaneus; blood-agar when trying for the streptococcus, meningococcus, gonococcus, Micrococcus catarrhalis, Micrococcus tetragenus, Micrococcus paratetragenus, pneumococcus, Bacillus influenzæ, or Friedländer's bacillus. Since the first group of organisms will also grow well on blood-agar, it is obvious that whenever we are in doubt we always use this medium.

When glucose agar is used, the bacteria are isolated by making several dilutions and plating.

When blood agar is used, the plates must be made at the time the blood is drawn, and subsequently inoculated by streaking the infected material over the surface by means of a bent glass rod or platinum wire. By both these methods isolated colonies are obtained, and pure cultures can be made by "fishing" out the different ones and making subcultures on the differential media necessary.

Pure cultures are essential because—

(1) The organisms must be identified in order to determine if they are pathogenic and also to know what temperature must be used to kill them.

(2) Different kinds of bacteria have different rates of growth on the same medium, and some organisms grow well together, while others do not, so in mixed cultures we do not obtain the organisms in the proper proportions.

(3) Almost all cultures from surface lesions are contaminated with saprophytic bacteria, which usually outgrow the pathogenic ones, and are, of course, useless for making a vaccine.

After pure cultures of the pathogenic organism or organisms are secured it is easy to prepare the vaccine. Only two precautions must be taken: (1) The pure cultures should not be contaminated during the process, and (2) the proper dosage must be obtained.

Method.—Several agar or blood-agar slants are inoculated with pure cultures of the organism from which the vaccine is to be made. The bacteria are spread over the entire surface and allowed to grow in the incubator. Twenty-four hours is usually sufficient. Salt solution (0.9 per cent. NaCl and 0.2 per cent. trikresol) is added to each tube, and the bacteria are washed down by rubbing the colonies free from the surface with a platinum wire. The heavy emulsion is then poured or pipeted into a sterile bottle containing glass pearls and thoroughly shaken, either by hand or, better, in a shaking machine. In this way the clumps are broken up and a uniform emulsion obtained. The mixture is then poured into sterile centrifuge tubes and whirled for a few minutes to remove any particles of culture-medium which may be present. The supernatant emulsion is pipeted into a sterile tube, and is now ready to be standardized.

Standardization of a vaccine means the determination of the number of bacteria in 1 c.c. of the mixture. There are several methods for doing this. Wright's is usually the most convenient. When using his technic, we compare the number of bacteria in the suspension with the number of red blood-corpuscles in human blood. We select some one whom we know to have about 5,000,000 cells in 1 c.mm. A capillary pipet with a mark about one inch from the tip and several clean glass slides are the only apparatus necessary. One or two volumes (one volume equals amount of fluid contained in the capillary tube between the mark and the tip) of potassium citrate are drawn up first, then exactly one volume of human blood, and, finally, one volume of the bacterial emulsion. The fluids are immediately expelled on a glass slide, then drawn up and expelled again. This is repeated several times to secure a uniform mixture. Small drops are placed on each of three or four slides, and ordinary blood-smears made. These are stained with methylene-blue or Wright's blood-stain. When examined, we see that the slide has numbers of bacteria distributed around the blood-cells.

A total count is then made of 10 oil-immersion fields of both the red blood-cells and the bacteria. From this count we can determine the number of bacteria per cubic centimeter of the suspension. For instance, if there are 300 bacteria and 500 red blood-cells in the fields counted, we say 300 : 500 : : x (the number of bacteria per cubic centimeter : 5,000,000. Therefore, x equals 3,000,000, the number of bacteria per cubic centimeter.

Since we have determined the concentration of the mixture, it is easy to dilute it so that we have 100,000,000, 200,000,000, or 500,000,000, in each cubic centimeter. For dilution we use the salt solution which contains 0.2 per cent. trikresol, so that we have the same concentration of the preservative in all dilutions of the finished product. The vaccine is now put up in 1 c.c. ampules, several for each dilution, and sealed.

These ampules are now sterilized by heating in a water-bath, usually at 58° to 60° C. for forty-five minutes.

They are tested for sterility by making cultures on appropriate media and by animal inoculation. If negative results are obtained, the vaccines are ready for use. They are labeled and kept in the ice-box until injected.

Vaccines Versus Sera.—In the treatment of infections it is necessary to decide whether the therapeutic sera or the bacterial vaccines are indicated. Vaccines, as stated above, are bacterial suspensions which, when injected, stimulate the organism to the formation of antibodies. Sera are fluids containing antibodies already formed, and are injected into the circulation to supply antibacterial elements without stimulating the body-cells to the production of these substances. Hence in the use of sera the antibodies formed in the body-cells of the horse are supplied to the patient, and a condition of passive immunity is established, lasting a few days or a few months.

There has been so much written on biologic therapy that it seems almost unnecessary to discuss the indications and contraindications for vaccines and sera. There is, however, some doubt in the minds of the average practitioners, and even some difference of opinion among the clinicians who use vaccines, just when the vaccines or sera are indicated.

Theoretically, sera should be given only in the toxemias, such as diphtheria or tetanus, but clinical experience has taught us to use sera in the acute general infections where the condition of the patient is such that the blood should be supplied with antibodies already formed, as in serum, rather than to tax the system by injecting bacterial vaccines, thus giving the organism the additional burden of making its own antibodies.

We believe that when the indication arises for the injection of vaccines, there is still one more question to be settled, namely, whether to inject the autogenous, the stock, or the mixed vaccines. We are convinced that the auto-vaccines are the agents par excellence of biologic therapy, and have made it a law that whenever it is possible to isolate a specific organism or specific organisms, the autogenous vaccines should always be made and given in a scientific, systematic, and fearless manner.

Why?

1. Their use is most logical and scientific, because they produce the specific antibodies most suitable and most effective for the individual case.

2. Clinically, they have given us the best results.

3. They rarely, if ever, produce harmful or annoying aftereffects.

4. Their preparation is simple, requires very little time, and is by no means expensive.

The most serious objection which can possibly be raised is that the time required in preparing the vaccines may greatly hazard the ultimate results. In rapidly progressing infections due to the more common organisms, it has been our custom to give a suitable stock vaccine as the primary dose, while the auto-vaccines are being prepared. *Stock vaccines* are indicated only in those cases where the suitable auto-vaccines cannot be readily obtained; for example, in the case of acne, where it requires from six to eight days for the full development of the acne bacillus culture. We have used *mixed vaccines*, both auto- and stock, where mixed infection has been clearly demonstrated. Their use does not necessarily signify the physician's inability to make an accurate bacteriologic diagnosis, nor need there be any hesitation on the part of the doctor because of the fact that they have been designated as unscientific. The fact is that there is a clear, positive, scientific indication for mixed vaccines in all mixed infections. We do not wish to infer that all bacteria found in a given infection must be injected with one puncture of the needle. Indeed, when treating acne, for instance, we have found that the best results are obtained by injecting the staphylococcus into one arm and the acne bacillus in the other at the same visit. One should not hope to cure a case of tuberculosis with a mixed infection by injecting tuberculin alone, nor can one expect to eradicate a cystitis due to the colon bacillus and the staphylococcus by injecting the colon bacillus or the staphylococcus alone. Again, we have found that, in a great number of cases, especially the old chronic or subacute arthritides, neither the primary focus nor the causative organism could be demonstrated. In these one must obviously resort to a mixed vaccine. Having had a large number of just such cases led us to the use of a mixed vaccine containing as its predominating organism the most likely cause of the infection. For example: In those cases where the primary focus, as nearly as could be determined from the clinical history, lay in the respiratory tract, we have had our bacteriologist prepare a vaccine containing pneumococci, staphylococci, and streptococci.

In the case of gastro-intestinal origin, we have used the Bacillus coli, staphylococci, and streptococci. Where the primary infection was in the aural cavity or the various skin lesions, in fact, any lesion bordering on a low-grade pyogenic infection, we have had excellent results from the injection of a home-made pyocyaneus and staphylococcus vaccine. This may not seem particularly scientific,—it may even seem empiric,—but it has given us results where all other types failed, and is justified on account of the clinical results awaiting a more exact and scientific procedure.

Clinical Considerations.—As you all know, sera, up to the present time, are said to be indicated only in the toxemias, and that vaccines are indicated in the bacteriemias.

In the last four years it has been our custom to give the antistreptococcic serum in all those cases of acute general streptococcic infections, acute streptococcic peritonitis, and in the extremely toxic cases of streptococcus infections of the extremities.

In a series of over 200 cases of this type each patient received from 10 to 40 c.c. of the streptolytic serum, and with a fall of the temperature and a subsidence of symptoms in a majority of the cases. You will understand, of course, that the sera are discontinued as soon as the auto-vaccines are prepared.

Our experience with the sera in scarlet fever has been limited to a few cases, but even in these we noticed beneficial results and never any untoward after-effects. In a surgical clinic we rarely meet with diphtheria, but in the few cases we have treated the results have been most gratifying. We have, however, noted in a few cases the symptoms of anaphylaxis which were so fully discussed before this Society two months ago.

The antitetanic serum is given in all cases of injury or gunshot wounds in which there is an open lacerated wound inflicted by machinery or implements which have been in contact with the dust or dirt. This is our routine practice, and has been used in a series of over 150 cases, and so proficient has been the prophylactic action of the serum that Dr. Murphy has not seen a case of tetanus originate in our hospital patients in seven years. The usual dose given is 1500 units. We give the serum therapeutically when occasion arises. I have had occasion to see in a distant city a case of tetanus in which the symptoms developed nineteen days after the primary infection. Repeated injections of the serum were given and the more violent symptoms subsided. The patient had no muscular twitching for ninety-six hours before his death.

Bacterial vaccines have been administered by us in all types and classes of cases such as are met in an extensive surgical practice.

Beginning at the lesions of the skin, we have treated cases of acne, furunculosis and carbunculosis, and infections of the lymphatic glands. Next we shall consider the lesions of the respiratory tract, as chronic rhinitis, asthma, hay-fever, bronchitis, empyema, and tuberculosis with mixed infection. Next chronic suppurations, including osteomyelitis and infected surgical wounds of all descriptions. Following this, chronic enteritis and kidney and bladder infections not associated with obstruction by foreign bodies. Then low-grade pyogenic or saprophytic infections, and last, but most extensive, has been our experience with vaccines in all types of acute and chronic joint infections.

Bacteriologically, our cases have included almost every variety of bacteria, with varying degrees of virulency. Most common among them we found the Staphylococcus aureus and albus, the streptococcus, pneumococcus, the Bacillus coli, and the Bacillus pyocyaneus.

Detailed Considerations.—The principles underlying the successful treatment of acne vulgaris are:

1. The determination and isolation of the causative organisms.
2. The knowledge and experience necessary for the determination of the dosage and the interval between inoculations.
3. The general condition of patient and the degree of virulency of the infective organism.

In the series of 15 cases treated by us we found that the Staphylococcus albus was present in 90 per cent. of the cases.

Theoretically, the acne bacillus should be present in every case. The difficulty of growing this bacillus is the principal obstacle to the successful treatment of acne vulgaris. This organism requires from five to ten days on the most suitable culture-media, one of which is the 1 per cent. oleic acid agar.

Another reason for failure is the fact that the dosage of the acne bacillus vaccine has been much too large. In all these cases it is also necessary to watch and modify the coagulability of the blood. If this is too rapid, the patient should have large doses of sodium citrate; on the other hand, if the coagulability is too slow and the patients do not improve immediately, it is necessary to give a number of 10-grain doses of the calcium lactate.

We do not encourage this type of cases, but many of them came to us for some surgical lesions. Several of them had made "the rounds," and finally appealed to us for the vaccine treatment.

In two of our cases all the lesions subsided after three or four injections, and have not returned. Some of the cases improved rapidly after the fifth or sixth injection. One patient was not improved at all.

The furuncles and carbuncles, of which we have treated 20 cases, have all made rapid and complete recovery. The autogenous vaccines should always be given if one wishes to obtain the best results. One of our cases, who had previously been treated with mixed stock vaccines over a period of seven months, responded immediately after three injections of autogenous vaccines. Eighty per cent. of these cases showed the Staphylococcus aureus.

Glandular infections, not tubercular, are due to the Staphylococcus aureus and albus, occasionally combined with the pneumococcus. Twenty-five cases of this type treated with autovaccines showed rapid improvement, and the majority of them went right on to ultimate complete recovery.

We have had one case of chronic rhinitis in a doctor, the most prominent symptom of which was a very annoying sneezing, which was treated with a mixed vaccine which contained the streptococcus and pneumococcus. The sneezing very materially improved after the fourth or fifth injection.

We have treated six cases of chronic asthma in which we were able to demonstrate the pneumococcus in one case, and the staphylococcus and pneumococcus combined in the other five. In one we noted an aggravation of the symptoms from ten to eighteen hours after the injection, but a marked improvement after the third dose. The other cases, one of which was not improved materially, did not show the reaction as did the first case. Another case of asthma, treated at the hospital under our direction, showed no pathogenic bacteria in any of the bronchial sections that were obtainable. We advised the use of a mixed pneumo- and staphylococcus vaccine. Later the intern informed me that the patient's condition was greatly improved and he has had no more attacks.

Last August we treated three cases of hay-fever with a mixed stock vaccine containing for the most part the streptococcus. Two of these cases did not find it necessary to change climate during the hay-fever season, as had been their custom for many years.

Four cases of purulent empyema with drainage were treated with auto-vaccines, resulting in a rapid diminution of the amount and odor of the discharge and ultimate permanent healing. Two patients had a green pus discharge, which delayed improvement for some weeks until a suitable autogenous vaccine of the Bacillus pyocyaneus could be prepared.

In tuberculosis with mixed infection we have made it a rule to use the vaccine, together with the tuberculin injections. The vaccines are given every four or five days, and the tuberculin once each week. Both injections should never be made on the same day, as that would interfere with the temperature reaction, which is the most important guide to the size of the tuberculin dosage.

Acute and chronic surgical infections of all types offer the largest field for vaccine therapy. Under this we include all cases which are primarily septic, incised or gunshot wounds, and osteomyelitis not associated with sequestra and not containing large cavities, pelvic infections, suppurations, appendicitis cases, etc. In a series of over 50 cases of this type we used the autogenous vaccines in about 90 per cent., and the mixed vaccines in 10 per cent. These cases, second to the pyocyaneus cases, give the most brilliant results. If the vaccines are properly made and the dosage and intervals properly graduated, this type should yield cures almost uniformly. The one reason for failure is the fact that the vaccines are not changed with sufficient frequency. A new vaccine should be made about every two weeks, to make sure that the organisms doing the most harm are incorporated in the vaccine. No culture can be transplanted more than two or three times without detrimental attenuation.

Another large field for biologic therapy is the kidney and bladder infections not associated with foreign-body obstruction or organic obstructions. The colon bacilli, staphylococci and streptococci, and often the pneumococci gain entrance to the urinary tract and cause marked frequency and often painful micturition. One of the most striking cases of this type came to the hospital late in October, 1911, suffering from frequent and painful urination, extending over a period of about ten months. After taking an accurate clinical history, it was found that this patient had suffered from a dysentery of several weeks' duration, and that her trouble dated from that time and became progressively worse. Cystoscopic examination showed no pus coming from either ureter, and the absence of a neoplasm or foreign body in the bladder. The culture from the urine made the diagnosis of cystitis due to the Bacilli coli and staphylococci. The auto-vaccines were given about every five days, and the patient made a rapid and complete recovery and has remained permanently well.

Another case, Mr. D., came to the office on December 11, 1911, giving the history of a continued illness for four months, with repeated daily attacks of severe pain deep in the perineum, associated with chills and high temperature, frequent urination (every quarter to one hour), and large quantities of pus in the urine. Prostate gland not perceptibly diseased. Culture of the urine showed Bacilli coli and staphylococci. Vaccines were made and the patient improved rapidly for several weeks, followed by a period of non-improvement for a few weeks. Another culture showed that all the staphylococci had disappeared, leaving a pure culture of Bacillus coli, from which a vaccine was made and injected in the usual way. This was in February, 1912. On April 1, 1912, Dr. Murphy made the following note: "The pains are all gone. Only one morning urination. The urine is absolutely normal." [Case has been permanently cured. July 2, 1913, patient reports himself perfectly well, with no evidence of return.]

Another patient, Mrs. G., suffering from a chronic cystitis, showed a bacillus not unlike the Bacillus pyocyaneus. The stock pyocyaneus vaccines were given at four-day intervals, and in a short time the pus-cells decreased and those that were present were greatly fragmented. This patient made a complete recovery, but had a recurrence from which she also recovered.

We have treated two cases of chronic enteritis, one of which was of tuberculous origin. This patient had from 10 to 20 bowel movements daily, most of them containing blood. The autovaccine was prepared, and after the first injection the blood ceased and the number of movements became less. The other case responded to none of the many vaccines given, and was later found to be a gas-bacillus infection.

In the last three years we have devoted our best efforts to the vaccine treatment of acute and chronic joint infections. This we have found the largest and at the same time the most difficult field of biologic therapy. The field is large, on account of the great number of cases with all types and varieties of joint infections that present themselves for treatment in Dr. Murphy's clinic. It is the most difficult type of cases from which to procure cultures for vaccines—*first*, on account of the large number of atria of infection; *second*, on account of the various types of organisms causing the arthritides; *third*, because some of the cases have their primary infection twenty or even thirty years before they present themselves for treatment; *fourth*, because it is often impossible to obtain a culture, even from the blood, which can be used in the preparation of an autogenous vaccine.

In our series of 90 cases of arthritis we have been able to distinguish three classes, as follows:

1. That type of deforming arthritis of gradual and insidious onset from which no organism can be obtained, either from the joints or from the blood-streams, nor from the secretions or excretions.

2. Those cases, mostly of the chronic variety, which had their origin in ancient infection of the mouth, pharynx, tonsils, respiratory tract, intestinal tract, from which we were unable to obtain any one specific organism at the time the patients came for treatment, but who yield to mixed vaccines.

3. Those patients with acute or chronic infections of the joints in which we are able to isolate a distinct organism or organisms which we believe to have been the cause of the trouble.

The *first* class is obviously the most difficult one to treat and yields the least satisfactory results. One such case, Mr. H., aged forty-nine, came to the office on September 20, 1912, on account of pain and stiffness in the shoulder-, hip-, and ankle-joints. The trouble began twenty-nine years ago, when patient was twenty years old. He began having a dull aching pain in the spine, which continued four years. The pain became much more severe, and invaded both knee-joints, both hips, and both shoulders. The spine began to curve forward. Patient had no chills, no fever, no sudden incapacity—just a gradual progressive involvement of all the joints. At the time of examination the spine was fixed from the tip of the coccyx to the atlas. Repeated vaccines were made from the urine, but there was very little improvement. A mixed stock vaccine showed but little reaction, and therefore very little improvement. All kinds of vaccines have been given, but the result thus far obtained has been very unsatisfactory.

Another more favorable case, M. C., aged twenty-six, came to the office on account of pain in the spine. His previous illnesses were negative, excepting an occasional coryza. When the patient was nineteen he began to have pain in the lumbar region of the spine, which was especially severe in the morning. One year later many vertebræ had become involved. No history of chills, no fever, no total incapacity at any time. We were unable to obtain any material for culture, and the question arose what to do for the patient. Working on the basis that his trouble was possibly due to a low-grade infection of some type, we concluded to give the patient injections of pyocyaneus vaccine. The first injection of 50,000,000 was given on November 14, 1911. On November 21st the note on history reads: "Patient feeling much better; joints are not so painful." One injection was given each week, and on December 19th the note on history was as follows: "Patient had headache and slight temperature after last injection. He feels like a new man. Even the motion in his neck has improved, and is almost painless." The injections of pyocyaneus vaccines were gradually increased, and three months after the beginning of treatment the patient was better than he had been for a whole year.

This case prompted us to use the pyocyaneus vaccine subsequently in many of the rheumatoid or deforming arthritis cases. The improvement in a majority has been slow, but progressive, and has given results which warrant its further trial.

The *second* type of joint cases in which we are not able to isolate any one specific organism is the one which has led us to the use of mixed vaccines. From the clinical history of a large majority of this variety of patients we find that they had a more acute onset, characterized by a chill, rise of temperature, pain and swelling of the joints, and a partial or total incapacity. At first one or several joints were involved, and later many or all the large joints of the body. Most of these cases, as they came to us, were of the chronic type, with the history of onset dating back many years. The majority had had originally some type of nose or throat infection, a smaller number had infections of the genito-urinary tract, and then follow the alveolar infections, gastric or intestinal disturbances, the infections of the pulmonary tract, furunculosis, and other lesions of the skin in the order named. The mixed vaccines used in these cases contained the staphylococcus and streptococcus, the colon bacillus, and the pneumococcus, combined in some form or other. These are now made at our laboratory by our bacteriologists, and are combined in such a way as to suit the particular case as nearly as possible, judging from its early clinical history. The pneumococci predominate in those cases where the ancient infection originated from the respiratory tract. Where the primary infection was in the gastro-intestinal tract or biliary passages, we use mixed colon bacillus vaccine.

One striking case of this class was a Mr. E., a farmer, who came to the hospital on account of painful and swollen knee-, wrist-, and ankle-joints. Originally, he had had some grip infection of the gastro-enteric type. Cultures from the secreta and excreta, and even a culture made from the blood when the patient came for treatment, revealed no definite pathogenic organism which could be thought responsible for the original infection. Mixed stock vaccines were injected in rapidly increasing doses, resulting in severe local and general reactions. The temperature after one injection reached 103° F. After two months of vaccine treatment combined with extension and one injection of formalin and glycerin into each joint this patient, who had been unable to dress or feed himself for six months, returned home to his duties.

When a severe reaction follows the administration of the vaccine, as exemplified in the case just cited, we are positive that at least one of the bacteria incorporated in the mixture is the proper organism to be given. When *no reaction* follows, we are sure that very little benefit can be expected from their further use.

This line of procedure may be subject to criticism, but clinically we have had some splendid results in cases that were totally incapacitated and almost hopelessly deformed, and it shows that many of the patients may be taken from the "scrap heap" and restored to usefulness. The profession has been self satisfied in making a diagnosis of "rheumatoid arthritis," and then turning the poor victim over to the fates. It is time that some effort was made in a therapeutic line.

Finally, we come to the third class of cases that border on the ideal for vaccine therapy, namely, the multiple joint infections, from which we were able to demonstrate the offending organism. Unfortunately, these cases are in the minority in our clinic, as we get mostly " derelicts." From these we always make autogenous vaccines and can hope to get good results.

One such case, a male, aged sixty-one, came to the office because of painful and swollen joints, with marked limitation of motion. His previous illnesses consisted mostly of painful and frequent urinations, for the relief of which he had had theprostate removed in 1907. In June, 1910, he began having dull, aching pains in both shoulder-joints, which continued until February, 1911, when the pain became much more severe, and the trouble then extended to the smaller joints of the feet. Next, the wrist- and finger-joints became involved. He came to the office on crutches and had difficulty in getting from the reception- to the examining-room. Examination of the urine showed 30 to 40 pus-cells to a ⅙ power microscopic field. Autogenous vaccines containing the Staphylococcus aureus were made and given, with rapidly increasing doses. In three months the patient left the hospital and resumed his work, walking without pain.

Another patient, Mrs. M., had for years been suffering from an extremely painful arthritis which had deformed both knee-joints and nearly all the joints of the upper extremities. The frequent and painful urinations from which this patient suffered and for which she had for years had bladder irrigations, led us to the possible cause of the trouble. The examination showed a pure culture of the Bacillus pyocyaneus. The vaccines, which are still being given, greatly improved the bladder condition after the second injection, and gave the patient increased joint symptoms the day following the injections. This patient is practically free from all pain in her joints, and should be able to resume her duties in her home.

Thus we have endeavored to tell you briefly of the earliest and original work which led to the use of the bacterial vaccines. We have attempted to define the various types of immunity and the practical application of the opsonic index. We have mentioned [briefly the indications for vaccines and sera, and gave a method for the preparation of bacterial vaccines. We recited those types of lesions in which the vaccines are applicable, including the lesions involving the respiratory, the gastro-enteric, and the urinary tracts, and the infections of the joints. We told you of our successes and cited our failures.

Conclusions.—By way of recapitulation, permit us to say:

1. That biologic therapy will in a short time, we believe, revolutionize the present-day treatment of many medical and surgical lesions, and particularly in the early acute infections.

2. That vaccines have failed in many instances to give the desired results because they were given in cases for which they were never intended.

3. That autogenous vaccines should always be given when it is possible to obtain them, but that there are cases in which there is a positive indication for mixed vaccines and occasional cases for stock vaccines.

4. That vaccines must not be expected to reconstruct tissues, organs, or joints that have been destroyed by known or unknown pathogenic organisms. That they are prophylactic against such destruction, and to be effective must be timely and intelligently administered.

5. That vaccines and sera, although now used as adjuncts in medical and surgical therapy, have done much to relieve suffering humanity, and have made producers of many who would otherwise have been burdens to society.

DISCUSSION

Dr. Charles L. Mix: I am sure that we are all very much indebted to Dr. Kreuscher for the presentation of the subject of vaccine therapy tonight. The subject matter has certainly been presented in a most complete and yet in an incomplete way—incomplete because you have not seen the cases cited. Seeing the cases that have been cured is the most important part of the whole procedure. The wonderful results which have occurred in the patients I have seen from time to time at Mercy Hospital is the thing that produces the greatest impression. To see a farmer who has been brought into the hospital unable to walk, walking around the halls after a few months' treatment with vaccine is certainly an extraordinary thing.

In Dr. Kreuscher's paper no mention was made of the injection of living bacteria into patients for the purpose of curing disease. He doubtless deliberately omitted all mention of this method of therapy, though I think that it should be brought to mind this evening merely to be condemned. At the present time much is being printed in the public papers concerning the efficacy of such a method of treatment, which from the point of view of bacteriology is, to say the least, open to severe criticism. So far as we know at the present time it is possible to attenuate living germs by growing them in suitable culture-media, but so soon as they are restored to other media their virulence returns. Granting that it is possible to attenuate a germ outside of the human body, what likelihood is there when it is reinjected into a human patient that it will not at once regain its former virulence? In my opinion it is extremely likely to do so, because the virulence is determined not so much by the germ itself as by the medium in which it is grown.

Turning now to the subject matter of Dr. Kreuscher's paper, the production of immunity by vaccines, I have this to say: The production of such immunity is certainly taking place in patients as the result of natural processes. We are daily watching the progressive immunization of our patients, oftentimes without being aware of the fact. Tuberculosis is a disease in which progressive immunization is excellently well exemplified. An individual suffering with this malady ordinarily has a temperature and a rapid pulse. The fever and rapid pulse are lowered from time to time after a period of rest, or, putting it in another way, after the patient has spent a whole night in bed he has a temperature in the morning which is either normal or subnormal, and his pulse-rate is correspondingly low. At night, after a day of activity, his temperature is above normal and his pulse-rate has risen. Why? Because during the day the patient's activities, his bodily motions, his increased vascular pressure, and lymphatic flow have led to an introduction into his body of the toxins of a large number of dead germs. From these toxins he gets his daily reaction, consisting of increased temperature and pulse-rate. Now, it is a noteworthy fact that if such a tuberculous individual is put absolutely at rest in bed for a few days to a few weeks his temperature will fall, frequently to normal. Just so soon as he gets up and moves about and exercises, his temperature will rise above normal. By his activities he has produced the same reaction which we are able to produce by our method of vaccine therapy.

Thus nature cures the case. The activity of the patient inoculates him with an unknown dose of the vaccine. He is inoculated, moreover, at uncertain and very likely too frequent intervals. Such auto-vaccination ought not to be permitted. Nature does many things well, and cures very many infectious diseases; but nature ought to be assisted whenever it is in the power of man to do so. Auto-vaccination ought, therefore, to be abolished, and in its stead the physician should step in and administer vaccines when they are necessary, and only when they are necessary. Thus the physician can take charge of the matter of immunization, controlling it absolutely. But in order to control it effectually he must be able to control the patient's activities as well as his vaccines, which means he must have the patient under his control in a hospital. A patient up and about does not improve so much as a patient at rest in bed.

As to the various kinds of vaccines generally made use of, the best results have been obtained, in my experience, in the use of the vaccines for tubercular, gonorrheal, staphylococcic, and Bacillus coli infections. In the streptococcic infections, from my own point of view, I do not think that the results have been very extraordinary, yet there are cases in which the streptococcic vaccines have assuredly given excellent results. Though Dr. Kreuscher did not mention the case in his report, I believe that he saved a scarlet-fever patient by the use of the streptococcic vaccine.

Another matter of great practical importance is that of the mixed infections. I have long since been accustomed to look upon the body as a piece of soil. Just as in a garden left to itself various sorts of weeds grow up and live rather amicably together, some perhaps being of positive assistance to the others, so also in our body germs live amicably together and at times in a state of symbiosis. Instances of this are so common as to require no mention, but reference may be made at least to the mixed infections in tuberculosis. Another element comes into play in the matter of these mixed infections, and that is the variations in the virulence among the different strains of the same species of bacterium. The idea, then, of combating these germs growing together in the body by a vaccine made from a single one of them does not seem to be fundamentally sound. Most assuredly a vaccine made from several strains of the same species ought to be more efficient than a vaccine made from a single germ; a vaccine made from two or three germs that are commonly associated in an individual for the same reason ought to be more potent than a vaccine made from a single one. Therefore, I believe in the so-called mixed vaccine therapy, the more so because many of these vaccines have done remarkably well at Mercy Hospital.

As to the type of case which was last mentioned by Dr. Kreuscher, the joint disturbances, a very careful history will often reward the examiner by giving to him the exact diagnosis, as Dr. Murphy may later on say. Bacteria behave in rather a characteristic manner when they get into the human organisms, and often betray their identity by their actions. This statement is explained by an illustration; for example, after an infection of the body a secondary focus may appear in a joint a definite period of time after the primary one. From the date of the original infection and the date of the appearance of the metastasis in the joint, the physician may be able to name the germ. Recall, for example, typhoid fever—when do the joint disturbances begin? In the third or fourth week. When do the joint disturbances after the period of acute infection in gonorrhea begin? In our experience the joint becomes infected only between the eighteenth and the twenty-second day. When do the joints become infected in the streptococcic infections? In our experience the second or third day. Each of the germs more commonly at fault in the joint infections has a definite time for infecting the joint after the primary infection. Again I repeat that if it is possible to obtain from the patient an exact history, it is oftentimes possible to come to a conclusion as to the exact germ which caused the original infection.

When a patient comes to consult the physician for a joint disturbance, it is usually one or more years after the original infection. Blood cultures and cultures made from the joint at this time are usually negative. A careful history may enable you to make a shrewd guess as to the organism which caused the trouble. Is there any way to find out the truth or falseness of your guess? We have found an excellent means. Whenever the right vaccine is selected, it produces a characteristic reaction. When Koch's tuberculin first came into use it was intended to be a therapeutic agent, but by reason of the fact that it produces a characteristic response in a tuberculous patient it became a means for diagnosis. Precisely the same thing was true of the gonorrheal vaccine. It was early noted that in cases of gonorrheal joints the gonorrheal vaccine produced a characteristic local reaction. If, therefore, you have made a correct estimate as to the identity of the germ at fault, you may confidently expect a local and constitutional reaction on the injection of a vaccine prepared from that germ. If no specific local and constitutional reaction is obtained, then it is not at all unlikely that the guess has been wrong.

These, then, are the points which I would like to emphasize: that the injection of living germs is contrary to good sense; that auto-vaccination is going on constantly in nature, but that it is time for it to be superseded by the intervention of the physician and the exact regulation of the amount of vaccine introduced; that mixed vaccine therapy is in accordance with common sense; that in chronic joint infections a careful history of the case will often enable the physician to make a correct diagnosis as to the germ at fault, and that such a diagnosis can be tested by the local and constitutional reactions obtained by the injections of a vaccine made from that particular germ.

DR. T. L. DAGG: Dr. Kreuscher's paper and the splendid elaboration by Dr. Mix leave very little for me to say in the way of expanding on the vaccine question. I am engaged only in making it up on the orders received from physicians.

There are certain points which have come up in my experience with the orders and gathered from the physicians' experiences. I believe the vaccines will be made in a much higher concentration rather than in a lower. When we began using the staphylococcus vaccines we had orders for 10,000,000, 20,000,000, and 50,000,000. Now we have orders calling for 100,000,000 and 150,000,000.

Now physicians are using it in that concentration, whereas before they claimed they had failures. That is also true of vaccines of the colon type. We are making vaccines of the colon type as high as 800,000,000, and I believe that we are going to make a higher concentration of the vaccine. We begin with a small dose and gradually lead up to the highest dose.

My experience with the pyocyaneus vaccines in ear diseases has been rather unfavorable. I am judging from the reports received back on autogenous vaccines that I have made. Of the vaccines in ear diseases, the staphylococcus have responded pretty well, the pyocyaneus not so well. The pneumococcus—I have scarcely any reports on the use of the pneumococcus vaccines in ear discharges. The most marked results are obtained with the colon autogenous vaccines in the cases of cystitis. The condition seems to respond very well to the vaccines, but in this case again we have to have a high concentration of the vaccine, beginning with a low dose and gradually leading up.

The method of giving the injections is important. I believe that giving vaccine oftener than every four or five days is a menace to the patient. The best period to use the vaccines, in my experience, is five to seven days.

There is another point of importance, and that is the length of time during which the autogenous vaccine seems to be efficient. We observed that in the treatment of an acne case where we were getting no response. We thought it might be due to the small dose or to the low concentration. I believe the efficiency of the vaccine expires after a period of ninety days, because if you get a new autogenous vaccine the symptoms will improve rapidly.

DR. J. L. MILLER: I was very much interested in hearing Dr. Kreuscher's paper, because he has reported a large number of cases showing a large variety of diseased conditions which he has treated with vaccines. It seems to me that of all these cases those patients with chronic arthritis are the most suitable for the study of the effect of any therapeutic agent, because we are all familiar with the course of these cases which have resisted practically all forms of treatment in the past. If the cases of chronic arthritis yield to the vaccine treatment, we have found a therapeutic agent of great value. It is always very difficult to watch the therapeutic effect of any material, and we must look on vaccine therapy at the present time as purely experimental and take it up with a feeling of the possible value which may come from this treatment in a great variety of troubles.

I think Dr. Kreuscher's statement regarding the use of autogenous vaccine is a very good one, because, at the present time, there are so many vaccines used and often the man manufacturing them does not know the nature of the infection. A stock vaccine might be perfectly proper provided we know the nature of the infective agent.

My own personal experience has been limited to the use of the vaccines in chronic joint infections and in acute infections of the bladder and kidney. In the colon bacillus infections, just as Dr. Dagg said, the results have been most striking, especially in the colon bacillus infection of the kidney.

Dr. L. Hektoen: I just have one little remark to make—not of very much consequence—on the use of streptococcic serum. It is very pertinent to state that a careful examination of certain antistreptococcic sera on the market shows that they contain antibodies to streptococcic opsonins. It has also been possible to demonstrate experimentally that they will cure streptococcic infections. We have also noticed in our cases of scarlet fever, with streptococcic infection, that the streptococcic serum at first injected under the muscles would permit a rapid absorption of the antibodies. It is to be remembered, as Dr. Mix said, that certain extremes may take place which we least expect. I remember an instance, three or four years ago, in a case of scarlet fever, where the streptococcic serum was suggested. Dr. Herrick suggested that we "wait until tomorrow." We did, and the patient was better. We did not give the streptococcic serum and the child recovered.

Just a word in reference to the gonococcic arthritis. I was surprised that the report of the paper did not make more comment on the gonorrheal arthritis, because it was my impression of a gonococcus infection of the joint that it was a most important form of chronic arthritis. Perhaps that is not true in the Mercy Hospital clinic.

As regards the use of mixed vaccines, it does seem to me that we ought to proceed very slowly and very carefully. It may be perfectly proper to use mixed vaccines, provided we are certain we have a mixed infection. I must compliment the reader of the paper on the great conservatism that was displayed throughout, and which very clearly pointed out that most of this work with the mixed vaccines is not experimental, but empirical.

There is one thing that I wish to emphasize particularly, and that is that these mixed vaccines should be prepared by the one who uses them. The doctor in charge knows more about the case than does some one in Detroit, Philadelphia, or Kansas City. The doctor can regulate these much better than if he used a commercial vaccine.

CLOSING DISCUSSION BY DR. MURPHY

Dr. Murphy: This is a topic that we have been very much interested in for a long period of time. It is a topic that is finally going to take hold of us and is going to rob us of some of our surgical practice. It is a topic that, I believe, admits of much hope for the future. It is a topic, as we now look on it, in its very initial stage, acting on the information that we are getting from clinical observations, that is moving us to feel cautiously each succeeding step in its advancement. It is with the hope of bringing out discussion and getting the experience of other men and gathering new light from their experiences that we present this clinical report tonight. It is not a paper. It is not in that sense that it is presented. It is just stating the facts to you as they come to us from clinical observation, and these facts we hope will lead us in time to do better work and to obtain better results than we are obtaining.

There are a number of things that we know little or nothing about concerning metastatic infections. There are a few points in which we see some rays of light in metastatic infections. First, the time at which metastases take place is just the opposite of the time at which they should take place. What do I mean? The metastases never take place when the infection is at its height of severity, but they take place in the early days of recrudescence, except in streptococcic infections. If one has an inoculation with a streptococcic infection, one can have the cocci transmitted through the blood and lymphatics, and taken out of the blood in the local metastatic point within a few hours after their admission. If we have admitted into the skin or beneath the skin a staphylococcus infection, it may not show a metastatic focus for days and weeks. All of us, men with gray hair or no hair, recall the time when we, as young men, saw pyemia in its rampant form. When did the patients get the pyemic chill? Not the day they got the infection, not two days, but eight days to three weeks after the infection; that is the time they have the pyemic chills except in the streptococcic variety. Here again with typhoid fever we have the typhoid bacilli in the blood within a few days after the infection; when do we get the metastases? Barely ever before the end of the third week. Why? As the bacilli are floating around in the blood so freely all that time, why is it that when they commence to diminish and disappear that we get the focal metastases? Again, the older men who lived in the days of virulent diphtheria, when you had six or eight deaths in a family in one epidemic, remember that when these patients died from mycoses (cardiac or cerebral) they did not die in the early days of the diphtheria. They did not die when the microörganisms were most prevalent. When did they die? Not when the gangrenous patch was on the throat, but at the time it was thrown off or shortly thereafter. That is when they died. That is when the myocarditis manifested itself; that is when the paralyses appeared: when the diphtheric membrane fell off and you said, "I will make my last visit tonight. My patient is cured." You were called before midnight on account of the rapid pulse, the acute delirium, or the "vivaciousness," the cyanosis, etc. The patient was dying and died before daylight. These conditions we did not then understand, but they are now being cleared up as the knowledge of bacteriology, metastases, and immunity is developing.

We know little or nothing of the etiology and modus operandi of metastases. We do know, however, the relative time at which they take place in the various arthritides. We know as precisely as we know when to expect the eruption in measles, scarlet fever, and typhoid, when to expect the arthritis after knowing the time of the primary distal infection. We know from the character and description of the primary infection what the probable cause of the metastatic infection was. When a careful analysis of these cases is made, one can invariably find out just where the primary infection was located, what the metastatic infection was, and then, from the etiology, form an idea of how destructive the pathologic lesion is going to be to that joint. Since 1904 we have been endeavoring to impress on the profession the etiologic relationship between the nasal, pharyngeal, cholecystic, dysenteric, alveolar, tonsillar, genito-urinary, furuncular, antrum, mastoid diseases, and the acute and subacute arthritides—so-called rheumatism—as phases of these infections. More recently the profession is accepting inflammation of the tonsil as an etiologic factor in arthritis; it is not at all as frequently a causal factor as some of the other infections mentioned above. We have named these in the order of frequency in which they appear to metastasize as arthritides. The connection between these inflammatory infections and the joint lesion is as closely related as the fever, sore throat, etc., of scarlet fever is to the rash, and is as easily recognized, when one learns of the "regular" period of time that must elapse between the primary infection and the joint manifestation of it, namely, the "period of metastasis." We have failed to connect the primary infection with the secondary joint manifestation for so many centuries, because of the length of time that must necessarily elapse between the two in the disease cycle.

A patient has a grip infection, which is one of the more common causes, and when the grip commences to get better, he suffers from "rheumatism" of his hip or elbow. He will tell you he has rheumatism, something has happened that on the tenth, eleventh, or twelfth day gave him a chill (exposure to cold, wet feet, draft, overeating, etc.). You know, from the clinical fact that his rheumatism began with a chill, that that man in all probability is going to have one or more joints ankylosed as a result of that infection. That is not new. Dear old Dr. Hollister, who gave me the only lecture on pathology I ever had when a student, described rheumatism in that lecture, described the differential diagnosis between pyemia and rheumatism. He said pyemia in joint infections began with a chill always; rheumatism did not. That is exactly true of the metastatic arthritides today. When they begin with a chill they result in an ankylosis and a destruction of one or more of the joints. That is a clinical fact. When they do not begin with a chill they rarely result in ankylosis; take, for instance, the infection we know the most about, in connection with the Neisserian types of arthritides. When do they metastasize? In our analysis of them 96 per cent. of the cases metastasize between the eighteenth and twenty-second day. You ask the individual patient what happened. He will say, "I was exposed to the rain"; "I stood in an open door"; "I stood in the front of the street-car." He did something, and did it between the eighteenth and the twenty-second day. How many times do patients do one or all of these things preceding the eighteenth or following the twenty-second day without gonorrheal metastases! That merely shows that there is a time in the cycle of these infective lesions when they are prone to metastasize under favorable conditions; that there is a definite time for the appearance of the metastases, as well as a definite place for their localization. Every one of the infections has its own definite time.

The next important item is that of treatment. In the treatment of these lesions we believe that probably the best results must and will come in the future and do come to us now from giving the vaccines in the early inflammatory stages of the disease, together with the mechanical aids at our command and the chemic neutralization of the local infection. In the chronic arthritides of many types, and in the acute arthritides, the laws of vaccine treatment correspond in every essential to the primary laws laid down by Pasteur on fermentation, and you cannot do good vaccine work unless you know the essential principles of his individual work.

COMMENTS BY DR. JOHN B. MURPHY AT THE CLINIC AT MERCY HOSPITAL ON WEDNESDAY, MAY 28, 1913

DR. MURPHY: Last night we had the proposition of Vaccines and Vaccine Treatment of Disease discussed before the South Side Branch of the Chicago Medical Society. In order that we may have some conception of the vaccine treatment in infection, it is necessary to give a little review of what has been done in the last fifty years in the treatment of infection. Let us ask ourselves what has been done by the profession in the last fifty years to stop or limit the destructive work of local infections. What has the medical man done to increase the resistance or eliminate infections in the last fifty years? What has he done to eliminate the local or constitutional infections, including the infective diseases, so called, as pneumonia, scarlet fever, or diphtheria? Let us divide it up and see how little has been done and how self-satisfied and how self-inflated we have been.

Some of us remember, when we began the treatment of infections with aconite and veratrum viride, the aconite and veratrum viride circulatory depressant period. This treatment was in vogue for twenty years or more, when some one advanced the theory that infections should be treated by stimulants. Then alcohol, strychnin, camphor, digitalis were heroically resorted to—the stimulant treatment of infections.

Then came the period in which we began to administer a few drugs that we believed would have an extremely inhibiting effect on the growth of the microörganisms in the blood or tissues, as salicylates, creosote, carbolic acid, antipyrin, antifebrin, aspirin, and now the chief goddess of deception—urotropin. And that is all we did until we came to take advantage of the processes of fermentation—not the mere killing of the microorganisms, but rendering the soil undesirable, a poor or nil culture-medium for the microörganisms. Then we began to supply the system with antibodies, first, in the form of a serum treatment for diphtheria; that is, that you put into the system antibodies, and these antibodies stop the growth of the diphtheria bacilli or neutralize the biotic or toxic ill effects of the organisms that are still in the blood.

Then came the era of serum treatment, which was the first real treatment that we had for infection, except the mechanical, which we are going to consider as the last proposition. In the administration of a serum the antibodies are formed in another animal or living organism and injected into the human, and are, therefore, hetero-antibodies or neutralizin, and contrast with vaccines, which produce their good effect through the stimulation of autodevelopment of antibodies, opsonins, of neutralizins.

Next we come to the management of infections by vaccine treatment. What do I mean by vaccines? The vaccine treatment is the stimulation of organs or cells in the body to the production of a material that either devours or neutralizes the infective flora or their toxins, and destroys the deleterious effect of the microorganisms. That is clear. All right. That is all we have done except the mechanical treatment.

The mechanical treatment of infection consists of relieving the tension on the products of infection and thus limiting the destruction of the infective material through opening and drainage.

All the above treatments of infection, except the sera and vaccines, are purely empirical. Opening a felon to stop the suppuration is an empirical process. We do not know *why* the opening and relief of tension stop the infection in a majority of cases, but we *do* know that it does. We do not take away the microorganisms that are in the tissue, abscess-wall, or cavity when we open them, still the mere opening causes a cessation of the destructive processes. In the vaccine treatment of infections we increase the resistance of the patient; we stimulate his auto-imaginary potency.

In a few diseases, as tetanus and diphtheria, the results have been astonishing; when timely administered, they have been all that we could desire. The vaccines are taken into the system and the body slowly develops the antibodies or opsonins. If all the microörganisms developed antibodies, then we could combat practically all the infections on that line, but when we take into account all the microörganisms, a great majority of which do not, so far as we know, develop antibodies, we cannot have any advanced definite idea of the effect on each system of a vaccine that has been injected. We should try to arrive at some conclusions from the clinical experience.

We come, then, to the last and most important matter which was a basis for the vaccine treatment, namely, that the microorganisms are destroyed by their own toxins, or by their prolonged habitat in a definite culture-medium, so that you may have microorganisms blighted or auto-destroyed if you concentrate them. That is exactly what happens. What happens with yeast ferment? It ferments for a certain time and then kills all the germs that are the medium of the ferment. It is through taking advantage of the basic principle in fermentation that we endeavor to put into the individual a vaccine that will develop new material able to make this system an uncomfortable resting-place or poor culture-medium for the microörganisms. Now we have in our systems, unfortunately, a number of varieties of structures, with varied circulations, with varied local resistances, and with varied classes of fermentation, which respond in different degrees to the material that you put into the blood, as is beautifully shown in the response to treatment of the different areas with spirochetic infections to arsenic and mercury. We have not learned as to how much of the vaccine is necessary to render the system a poor culture-medium. To put it in the words of Wright, "how much of the vaccine you should give each patient to produce a superlative opsonic reaction against these microörganisms." Knowing so little as we do of the biology of the microörganisms, all the work in that way must be on an empirical basis for the present.

We have built up a very complicated system of medicine, which is becoming more and more complicated each day. We have lost sight of the principal, every-day things, namely, that infections represent a very large percentage of the total of diseases, and in the last fifty years we have given for this class vasomotor stimulants, vasomotor depressants, antiseptics and antimicrobics, and finally injections of sera and vaccines, and to these we have added mechanical means. Five things: we can sum them all up in only five steps made in fifty years. We have not been holding our ultrascientific investigations close enough to the patient, which shows that we have been drifting away and blocking the purpose for which this great complicated, scientific structure of medical education and medical practice was effected, namely, the purpose of doing most for the patient individually and collectively.

FURTHER COMMENTS BY DR. JOHN B. MURPHY IN THE COURSE OF THE CLINICS AT MERCY HOSPITAL ON WEDNESDAY, JUNE 4, 1913

DR. MURPHY: Just how vaccines act we do not know. We know little of immunity except the empirical fact of immunity. We know this, that in joint infections, specifically and in infections at large, aside from the mere mechanical aids, we have done nothing whatever to relieve these infections in the last fifty years, notwithstanding our inflated advancements. If you think that statement is not correct, just tell me one infection, other than those affected by vaccines or sera, that we have done anything for in fifty years. Relieving the tension on pus products and relieving the tension on the infective products has been the one advance that has been made; *relieving it timely* has been the last step in the advancement, and that step is being made by the profession at a sub-snail pace.

Today our pyocyaneus infections are cured by one or two injections of an autogenous pyocyaneus vaccine; many times cured by the ordinary stock vaccine of pyocyaneus; second, infections of the urinary tract due to the colon bacillus, where there is no foreign body and where there is no obstruction, have also been cured by injections of the auto-colon bacillus vaccine. In these two instances you have the cure of the infections by the vaccines with great regularity. From these two central or developing points we are endeavoring to widen the zone of vaccine therapy on the only basis we know of for widening it, which is the empirical basis through clinical observation.

Let me talk about vaccines and sera. The serotherapy of diphtheria is an unqualified success. The serotherapy of tetanus, as a prophylactic means, is an unqualified success. The serum treatment, if commenced early, for rabies is an unqualified success.

Then the influence dwindles and dwindles as you come down from that.

What is the difference between vaccines and sera? A vaccine is a material that is injected into the body, made from specific germs, to increase by its influence the antibodies which that vaccine is believed to produce. Is that clear? A serum is what? A serum is the injection into the system of antibodies. The sera immunize through their own antibodies from another organism. Sera do not depend on the resistance of the individual organism into which it is injected to produce antibodies. You put in antibodies or antibody influence. One is immunity protection from any other organisms; the other is stimulation of the patient's individual or auto-immunizing forces, wherever they may be located or however they may be called into activity. Whether it is an opsonin or an antibody we often do not know. It does not matter to the patient. The clinical fact is that we want to induce the effect that is produced by the injection of the vaccine.

In those cases in which we have been able to locate the individual type of the infective organisms that produces the metastases we realize very decided improvement, and particularly from the autogenous variety. First, every man can make his own vaccines if he is versed just a little in the technic of that work. Second, we get a lesser response from the stock than from the auto-vaccines, even including the pyocyaneus. Third, some of the organisms have no antibodies that we know of up to this time. Just recently it has been shown that we could produce a vaccine from actinomycosis. It did not look as though the actinomycoses would produce antibodies, but in the recent English literature I read of an autogenous vaccine made from actinomycosis pus, thus showing that the range is constantly increasing. It is not advancing with the profession as a means of treatment in the same way that a drug treatment would advance. Why? Because it involves with each case an individualization, and I would say that that has been a shortcoming of the medical profession in not individualizing the treatment of a case. We all remember how dear old Dr. J. Adams Allen used to dwell on this point in his lectures, that you should not treat the name of the disease, but treat the patient that has the disease. It has been hard for us to work effectively with vaccines because the *stages* of the disease, the *activity* of the disease, the *tissues* involved in the disease, the *individual resistance* of the patient, must control the dosage. There is no *average* dose—no average line that one can strike. There is no place where that is more beautifully illustrated than in the administration of tuberculin, because with tuberculin you can give one patient a thousand times the dose you give another, and the large dose will be too small and the small dose too large to produce the best opsonic resistance to the tuberculosis. These are the basic questions that we are endeavoring to work out in connection with the metastatic infections of the joint, and as our cases are mostly advance ones, they are difficult problems, but we are confident some one will soon take up the new treatment with a large clinical material and more rapid results will obtain.

THE BLOOD-SUPPLY IN AND AROUND THE JOINTS

THE accompanying series of skiagrams, showing the blood-supply in and around many of the important joints, were made in the *X*-ray Department of Dr. John B. Murphy's Clinic by Dr. George W. Hochrein, who does all the radiographic work for Dr. Murphy. The idea of these skiagrams suggested by Dr. Murphy had for its object the accurate determination of the blood-supply and its relations to the various joints, and are a great aid in determining the position of the flap pedicle used in arthroplasties. Professor Ranson, of the Department of Anatomy of the Northwestern University Medical School, prepared the cadavers by injecting the vessels with a red-lead emulsion. Each vessel and its branches are numbered and named according to the old and new nomenclatures by Mr. Sumner Koch, one of Professor Ranson's assistants in the Department of Anatomy. The whole series comprises a unique and valuable collection in applied anatomy, and is used at the clinics of Dr. Murphy in his work on the bones and joints.

FIG. 129.—THE LEFT SHOULDER-JOINT

1. Subclavian artery (A. subclavia)
    Thyroid axis (Truncus thyreocervicalis). Not visible
3. Superficial cervical artery (A. cervicalis superficialis)
4. Suprascapular artery (A. transversa scapulæ)
2. Transverse cervical artery (A. transversa colli)
5. Axillary artery (A. axillaris)
14. Acromiothoracic artery (A. thoracoacromialis)
7. Long thoracic artery (A. thoracalis lateralis)
8. Subscapular artery (A. subscapularis)
9. Dorsalis scapulæ artery (A. circumflexa scapulæ)
10. Anterior circumflex artery (A. circumflexa humeri anterior)
11. Posterior circumflex artery (A. circumflexa humeri posterior)
12. Brachial artery (A. brachialis)
13. Superior profunda artery (A. profunda brachii)

The dark area over the chest-wall was caused by an extravasation of injection-fluid under cover of the pectoralis major, from pectoral branches of the acromiothoracic artery.

The numeral "6" represents the anastomosis on the dorsum of the scapula. The anastomoses about the acromion and about the surgical neck of the humerus can be clearly distinguished.

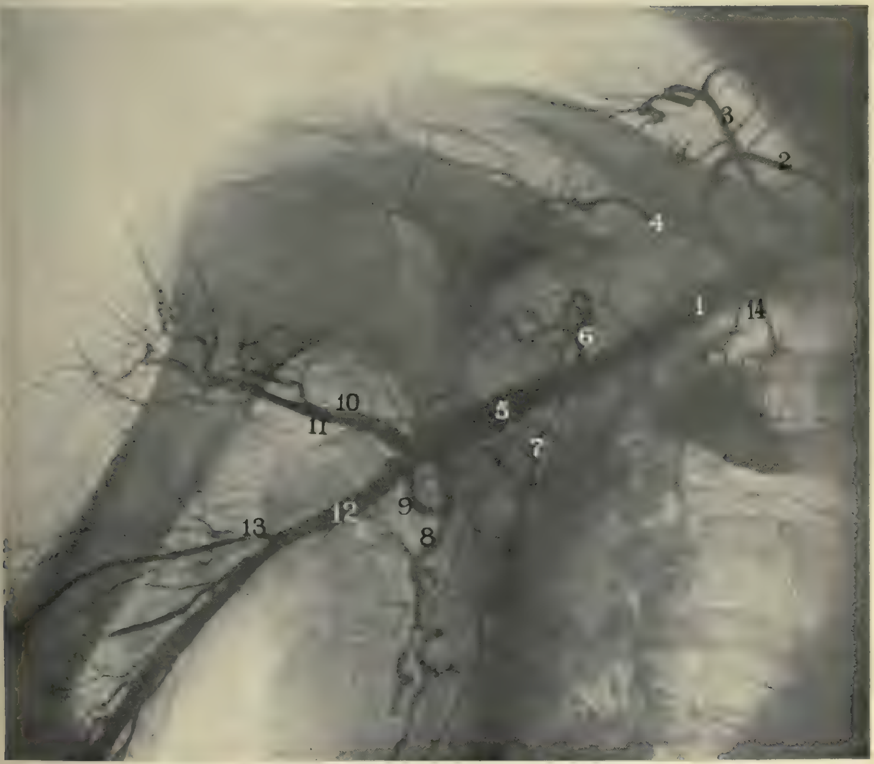

Fig. 129.—The left shoulder-joint.

Fig. 130.—The Right Elbow-joint (Lateral View)

1. Brachial artery (A. brachialis)
2. Superior profunda artery (A. profunda brachii)
3. Inferior profunda artery (A. collateralis ulnaris superior)
4. Anastomotica magna artery (A. collateralis ulnaris inferior)
5. Radial artery (A. radialis)
6. Radial recurrent artery (A. recurrens radialis)
7. Ulnar artery (A. ulnaris)
8. Anterior and posterior ulnar recurrent arteries (Aa. recurrentes ulnares), by common origin
Common interosseous artery (A. interossea communis)
9. Anterior interosseous artery (A. interossea volaris)
10. Posterior interosseous artery (A. interossea dorsalis)
11. Interosseous recurrent artery (A. interossea recurrens)

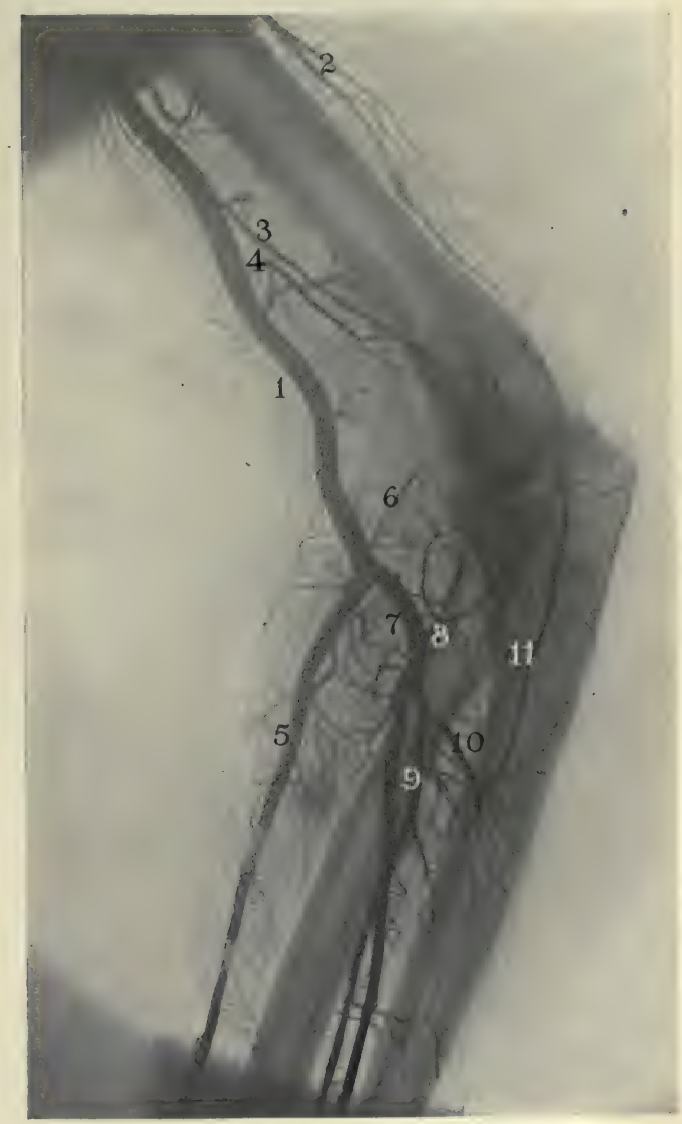

Fig. 130.—The right elbow-joint (lateral view).

Fig. 131.—The Right Elbow-joint (Dorsovolar View)

1. Brachial artery (A. brachialis)
2. Superior profunda artery (A. profunda brachii)
3. Inferior profunda artery (A. collateralis ulnaris superior)
4. Anastomotica magna artery (A. collateralis ulnaris inferior)
5. Radial artery (A. radialis)
6. Radial recurrent artery (A. recurrens radialis)
7. Ulnar artery (A. ulnaris)
8. Anterior and posterior ulnar recurrent arteries (Aa. recurrentes ulnares), by common origin
Common interosseous artery (A. interossea communis)
9. Anterior interosseous artery (A. interossea volaris)
10. Posterior interosseous artery (A. interossea dorsalis)
11. Interosseous recurrent artery (A. interossea recurrens)

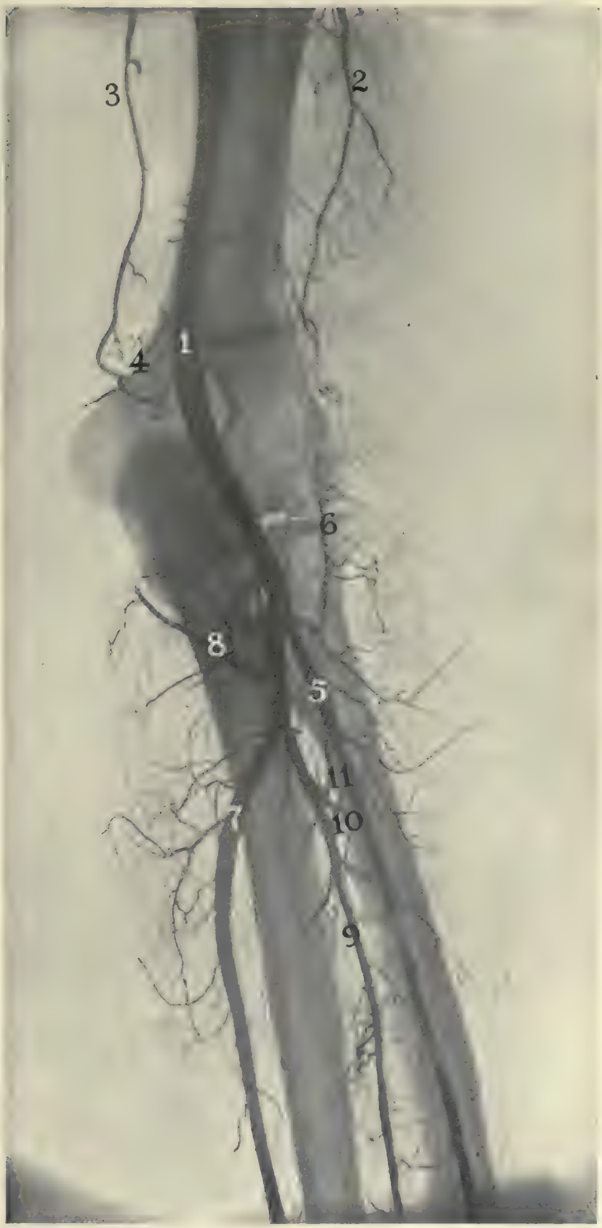

Fig. 131.—The right elbow-joint (dorsovolar view).

FIG. 132.—THE RIGHT FOREARM (LATERAL VIEW)

1. Ulnar artery (A. ulnaris)
    2. Anterior interosseous artery (A. interossea volaris)
    3. Anterior ulnar carpal artery (Ramus carpeus volaris)
    4. Posterior ulnar carpal artery (Ramus carpeus dorsalis)
    5. Deep palmar artery (Ramus volaris profundus)
6. Radial artery (A. radialis)
    7. Superficial volar artery (Ramus volaris superficialis)
    8. Posterior radial carpal artery (Ramus carpeus dorsalis)

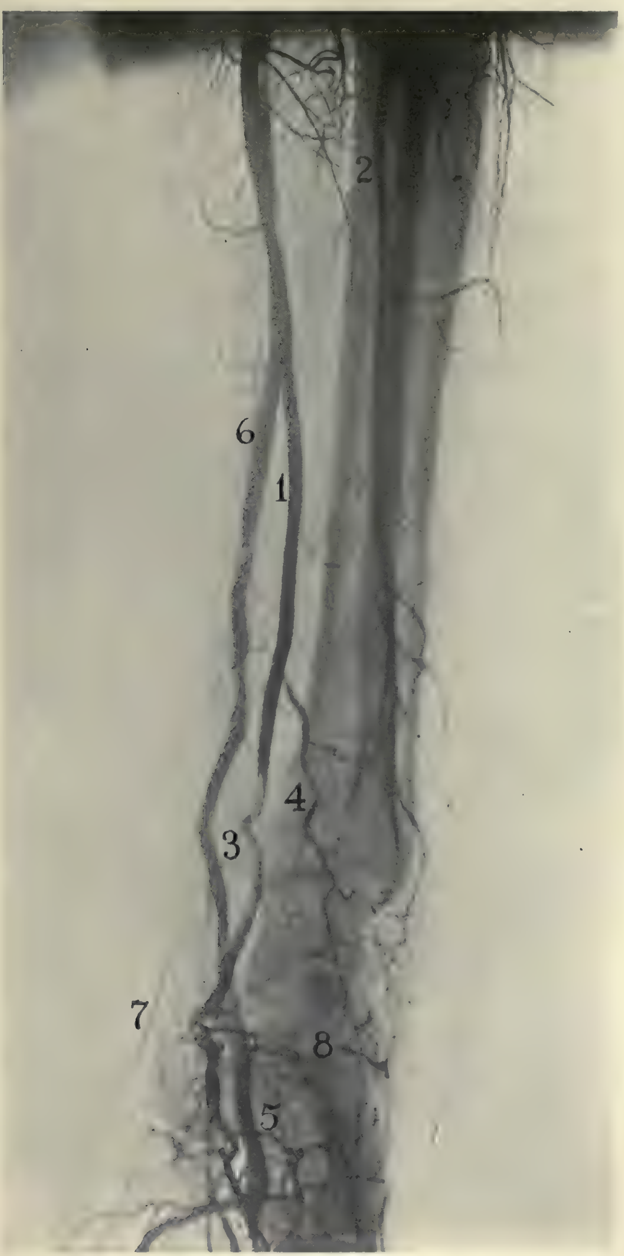

Fig. 132.—Right forearm (lateral view).

FIG. 133.—THE LEFT FOREARM AND HAND

1. Ulnar artery (A. ulnaris)
    2. Anterior interosseous artery (A. interossea volaris)
    3. Posterior interosseous artery (A. interossea dorsalis)
    4. Posterior ulnar carpal artery (Ramus carpeus dorsalis)
    5. Deep palmar artery (Ramus volaris profundus)
    6. Superficial palmar arch (Arcus volaris superficialis)
7. Radial artery (A. radialis)
    8. Anterior radial carpal artery (Ramus carpeus volaris)
    9. Superficial volar artery (Ramus volaris superficialis)
    10. Posterior radial carpal artery (Ramus carpus dorsalis)
    11. Dorsalis pollicis artery (Ramus dorsalis pollicis)
    12. Dorsalis indicis artery (Ramus dorsalis indicis)
    13. First volar metacarpal artery (A. metacarpea volaris I), dividing into the princeps pollicis artery (A. princeps pollicis) and the radialis indicis artery (A. volaris indicis radialis)
    14. Deep palmar arch (Arcus volaris profundus)

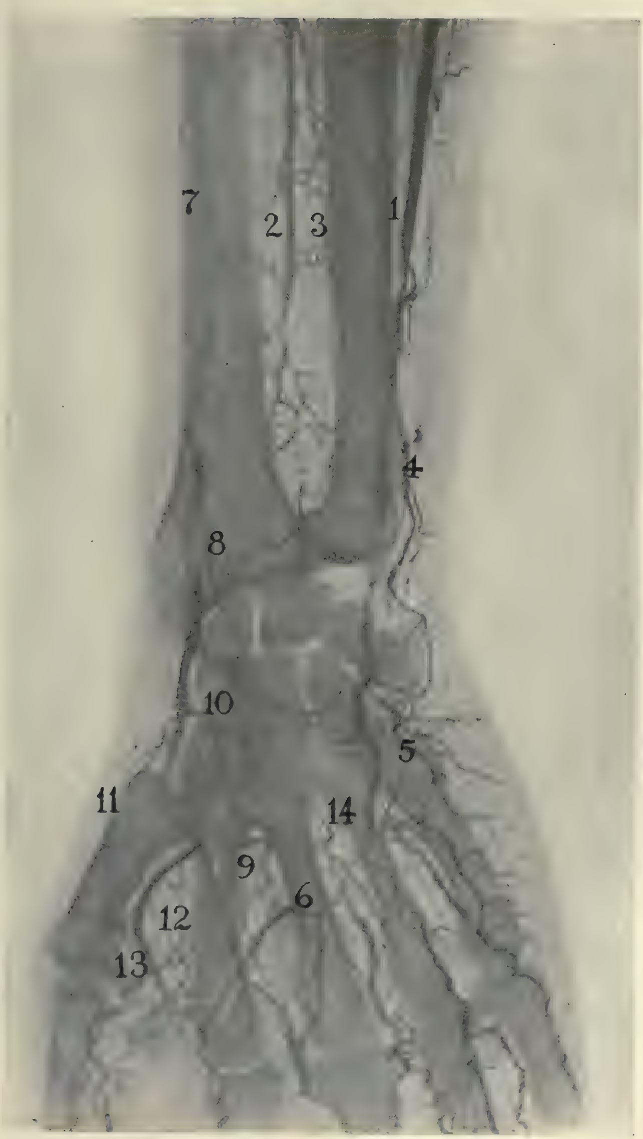

Fig. 133.—The left forearm and hand.

Fig. 134.—The Left Hip-joint

1. Spermatic artery (A. spermatica interna)
2. Common iliac artery (A. iliaca communis)
3. Internal iliac artery (A. hypogastrica)
    5. Iliolumbar artery (A. iliolumbalis)
    6. Gluteal artery (A. glutæa superior)
    7. Obturator artery (A. obturatoria)
    8. Sciatic artery (A. glutæa inferior)
    9. Internal pudic artery (A. pudenda interna)
4. External iliac artery (A. iliaca externa)
10. Femoral artery (A. femoralis)
    11. Deep femoral artery (A. profunda femoris)
        12. External circumflex artery (A. circumflexa femoris lateralis)
        13. Internal circumflex artery (A. circumflexa femoris medialis)
        14. First perforating artery (A. perforans prima)

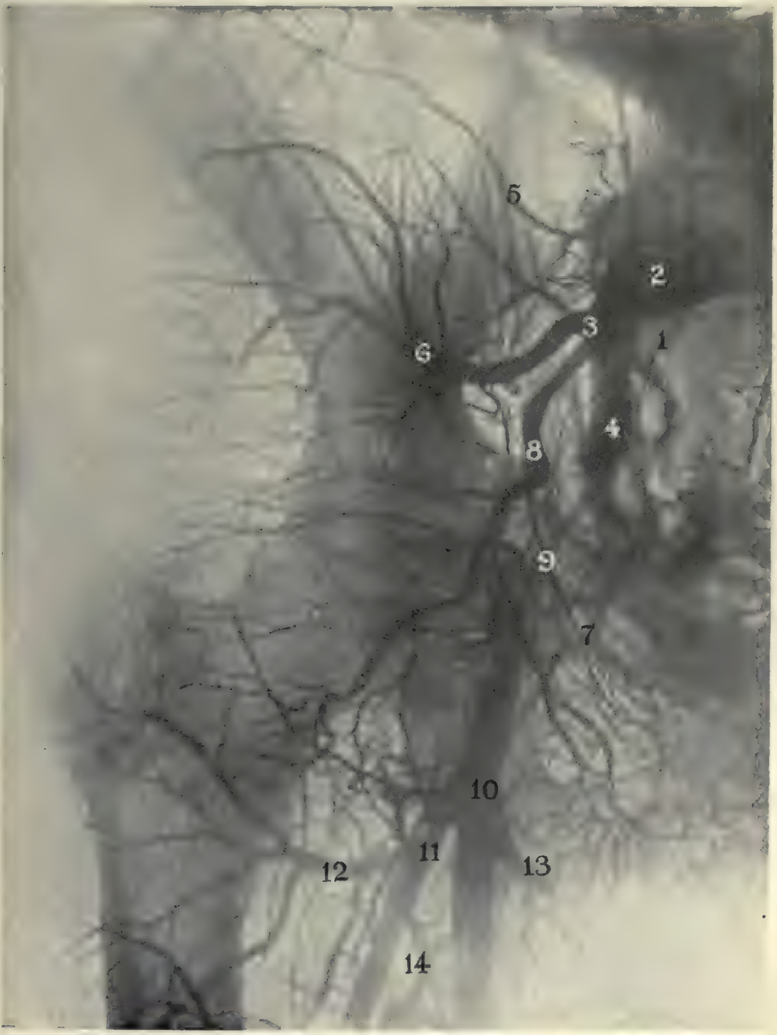

Fig. 134.—The left hip-joint.

Fig. 135.—The Right Knee-joint (Anteroposterior View)

2. Anastomotica magna artery (A. genu suprema), from deep femoral
1. Popliteal artery (A. poplitea)
   3. External superior articular artery (A. genu superior lateralis)
   4. Internal superior articular artery (A. genu superior medialis)
   5. Azygos articular artery (A. genu media)
   6. External inferior articular artery (A. genu inferior lateralis)
   7. Internal inferior articular artery (A. genu inferior medialis)
   8. Sural arteries (Aa. surales)
9. Posterior tibial artery (A. tibialis posterior)
10. Anterior tibial artery (A. tibialis anterior)
   11. Posterior tibial recurrent artery (A. recurrens tibialis posterior)
   12. Anterior tibial recurrent artery (A. recurrens tibialis anterior)

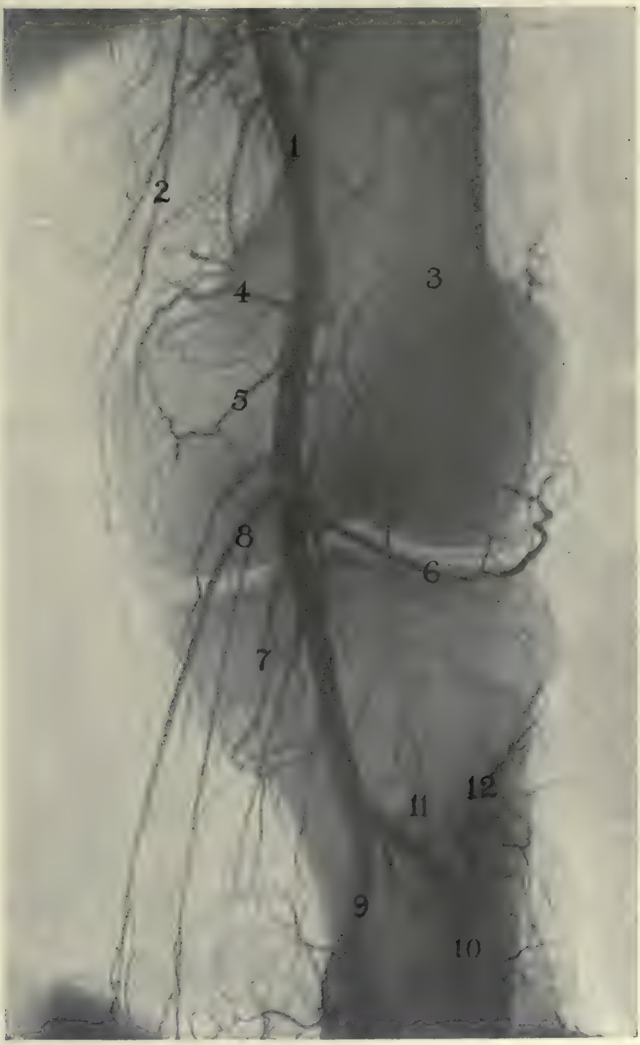

Fig. 135.—The right knee-joint (anteroposterior view).

Fig. 136.—The Right Knee-joint (Lateral View)

2. Anastomotica magna artery (A. genu suprema), from deep femoral
1. Popliteal artery (A. poplitea)
    3. External superior articular artery (A. genu superior lateralis)
    4. Internal superior articular artery (A. genu superior medialis)
    6. External inferior articular artery (A. genu inferior lateralis)
    7. Internal inferior articular artery (A. genu inferior medialis)
    8. Sural arteries (Aa. surales)
9. Posterior tibial artery (A. tibialis posterior)
10. Anterior tibial artery (A. tibialis anterior)
    11. Posterior tibial recurrent artery (A. recurrens tibialis posterior)
    12. Anterior tibial recurrent artery (A. recurrens tibialis anterior)

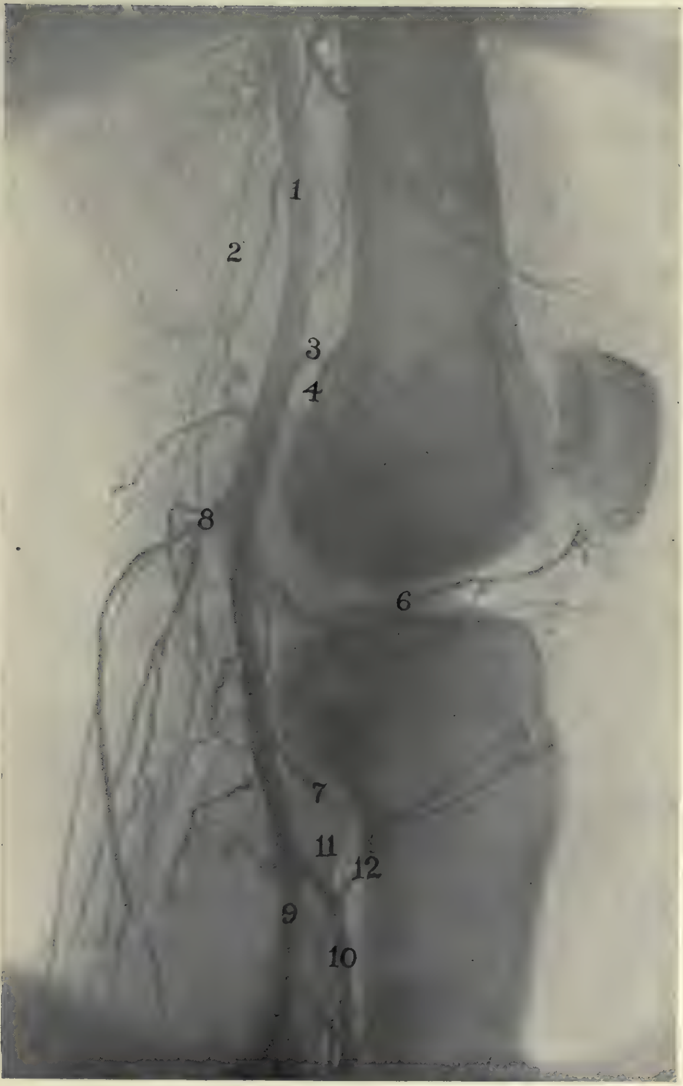

Fig. 136.—The right knee-joint (lateral view).

FIG. 137.—THE RIGHT ANKLE-JOINT

1. Posterior tibial artery (A. tibialis posterior)
    2 Peroneal artery (A. peronæa)
        3. Communicating artery (Ramus communicans)
        4. Anterior peroneal artery (Ramus perforans)
        5. Posterior peroneal artery (A. malleolaris posterior lateralis)
        6. Communicating artery, joining termination of peroneal artery and posterior tibial artery
7. External plantar artery (A. plantaris lateralis)
8. Internal plantar artery (A. plantaris medialis)
10. Anterior tibial artery (A. tibialis anterior)
12. Dorsalis pedis artery (A. dorsalis pedis)
    11. External tarsal artery (A. tarsea lateralis)

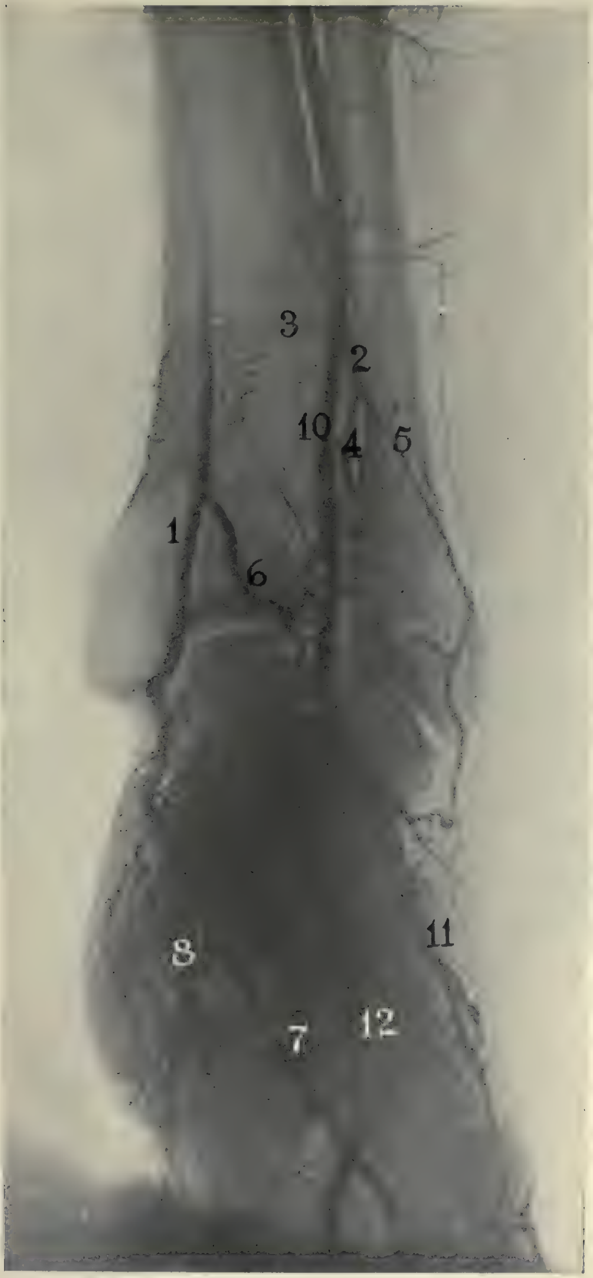

Fig. 137.—The right ankle-joint.

Fig. 138.—The Right Foot

1. Posterior tibial artery (A. tibialis posterior)
2. Peroneal artery (A. peronæa)
3. Communicating artery (R. communicans), connecting posterior tibial and peroneal arteries
4. Anterior peroneal artery (R. perforans)
5. Posterior peroneal artery (A. malleolaris posterior lateralis)
6. Internal malleolar artery (A. malleolaris posterior medialis)
7. Internal calcaneal arteries (Rami calcanei mediales)
8. External plantar artery (A. plantaris lateralis)
9. Calcaneal arteries (Rami calcanei)
10. Plantar arch, giving off the plantar interosseous arteries (Aa. metatarseæ plantares)
11. Internal plantar artery (A. plantaris medialis), dividing into two branches at the anterior border of the calcaneus
12. Anterior tibial artery (A. tibialis anterior)
13. Internal malleolar artery (A. malleolaris anterior medialis)
14. External malleolar artery (A. malleolaris anterior lateralis)
15. Dorsalis pedis artery (A. dorsalis pedis)
16. External tarsal artery (A. tarsea lateralis)
17. Dorsal anastomosis (Rete dorsalis pedis)
18. Metatarsal artery (A. arcuata)

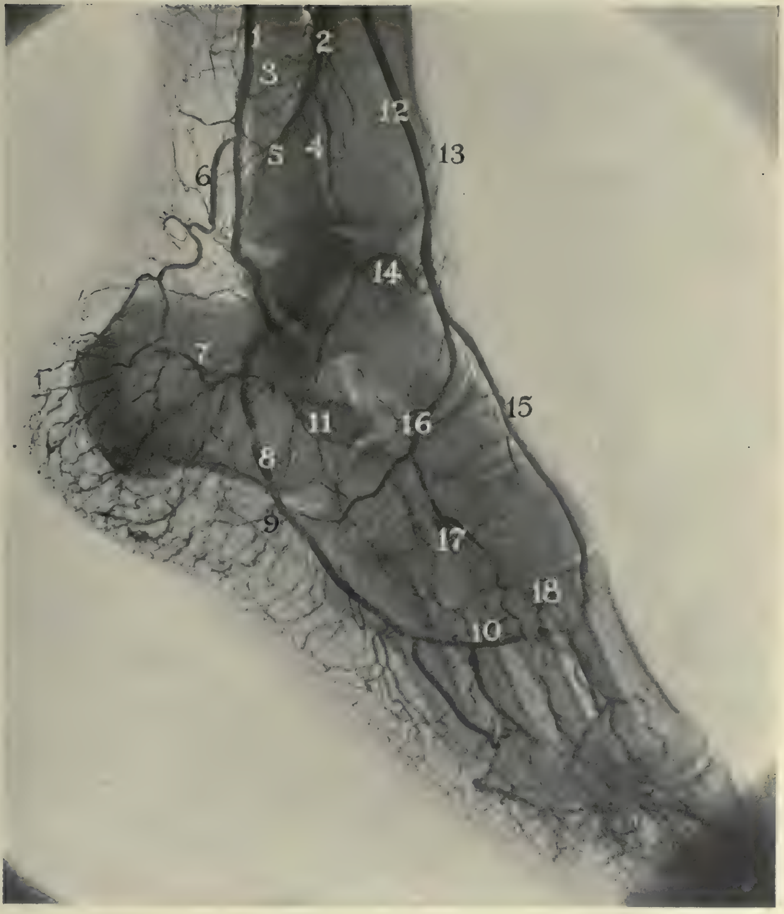

Fig. 138.—The right foot.

URETHRORECTAL FISTULA

Following Perineal Operation for Vesical Calculus

HISTORY

PATIENT, male, single, aged forty-four, comes to hospital on account of frequent and painful urination and a leaking sinus in the perineum from which urine escapes on urination.

When the patient was six or seven years old he had some trouble on urination. He does not remember what it was. An operation was performed through the perineal route and a stone was removed from his bladder. The wound closed. From that time on, however, he always had a burning pain on urination and the urine was always cloudy. When about eighteen years old the patient began to have a great deal of trouble with urination, having to urinate 10 to 15 times a day, and 4 or 5 times at night. He would pass a small amount of pus at the beginning and end of urination, and void only a small amount of urine at a time. He also had burning and pain on urination. At this time he also noticed that when the pain was very severe he would void urine through the rectum when he urinated. At twenty-five he was very sick, and lost a great deal of weight. He was operated on again for stone by the suprapubic route. No stone was found. The bladder was drained for two months. Examination at this time disclosed a fistula between the bladder and the rectum. No repair of the fistula was made. After this second operation he felt fairly well for eight or nine years, although urinations were frequent. He still voided pus and had a burning sensation on urination. At thirty-seven he was operated on for repair of the fistula. He was in the hospital four weeks. For the next three and one-half years he had very little trouble. He had no smarting or burning on urination, no pus was voided, except when he drank very little water. He urinated frequently,—10 or 15 times a day,—but voided large amounts of urine each time. He had no leakage of urine into the rectum. During this time his bowels were regular, whereas previously they had given him a great deal of trouble on account of passing urine through the rectum. His general health was good and he gained in weight.

The latter part of November, 1912, the patient again began passing urine through the rectum, and he also began to have pain on urination, voiding pus at the beginning and end of urination. The urine was cloudy. He also had to urinate frequently at night. During the last two months he lost 10 or 15 pounds in weight. Three weeks ago, March 24th, he had a chill, followed by a temperature of 104° F.; no pain, but nausea and vomiting and diarrhea. Patient always has to urinate once or twice at night. He has never passed bright-red blood in the urine. Venereal infection denied. Past history negative.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 15, 1913): He passed urine through his rectum at the time of urination, but the history does not state that he passed urine with the bowel movements, except when they were associated with urination. One would infer from this that the opening is from the urethra into the rectum and not from the bladder into the rectum; that is what would be indicated by that story. What do you mean by "fairly well for nine years"?

Intern: He gained in weight.

Dr. Murphy: Is the urine coming through the rectum now?

Intern: Yes.

Dr. Murphy: When?

Intern: Only when he is urinating.

Dr. Murphy: What we want to know is whether there is a communication between the bladder and the rectum or a communication between the urethra and the rectum. How long has he had it? If this fistula has been closed for nine years, why does it break open now? We filled his bladder with methylene-blue solution and retained the catheter in the urethra. None of the solution appeared in the rectum, notwithstanding the overdistention of the bladder. When the catheter was removed and he endeavored to urinate, then the methylene-blue solution passed into the rectum and was voided from it. This showed conclusively that there was no communication between the bladder and the rectum. Why did he have the frequent urination and the cystitis? Because he had a diverticulum and an infection in the prostatic urethra which kept up a continuous irritation.

A fistula leading from the rectum to the bladder in males is a most difficult proposition to handle surgically. What is the prognosis? The chances are four to one that we will not succeed in getting the fistula to close. That is a very unpleasant proposition for the patient, but at the same time it is the truth. Has he an infection of the bladder from the rectum? There is no history of his having passed gas from the bladder. When there is a communication between the bladder and the rectum, the rule is that they pass gas from the bladder. As he urinates the urine goes from the bladder into the urethra and from the urethra into the rectum.

We are unable to pass a sound into the bladder. I do not like that. We can pass a soft-rubber catheter. There is the urine. That urine may be from the rectum, but the chances are it is from the bladder. It is clear, amber-colored, and devoid of odor. We will now place the patient in the lithotomy position.

There is the transverse scar in the perineum from the previous operation. It is all cicatricial tissue here. This is one of the sequences of the old-style operation for stone in the bladder that was done on this man when he was about seven years old. It was a very serious operation. The present suprapubic operation came into vogue after that time. We will make a transverse incision in the perineum and dissect out that old scar. The finger in the rectum and the catheter in the bladder will guide us in this work.

There is the wall of the rectum, and there is the urethra. There is a point of approximation between the urethra and the rectum. That is where the urine passes from the former into the latter when he urinates. We will inject methylene-blue to see whether it communicates with the rectum beyond this urethral fistula. It does not. I will now dissect out this fistulous tract. There is much scar tissue near the urethra where the fistula was. There is the urethra. We will dissect this fistulous tract free from the urethra by making a transverse incision into the latter. This wound will be closed longitudinally with catgut, turning the edges of the incision out. The fistulous tract is then dissected free from the rectum, and the rectum is freed from the bladder for an inch above the fistula, so that an entropion of the rectal portion of the fistula can be made with two rows of sutures.

The external incision will be closed with deep catgut sutures which will obliterate all dead spaces. Silkworm-gut tension sutures and horsehair skin sutures will complete the closure. The catheter will remain in the bladder for ten days, and a rectal tube will be inserted. This tube will not be removed for at least a week.

This is the only feasible operation to perform in these cases, but the result is by no means always a satisfactory one. Not more than 25 per cent. of these cases result in a cure. That is unfortunate, but true.

Let the record show that a tubeful of methylene-blue was injected and showed that there was no leakage into the rectum or from the perineum. A tube was left in the rectum, and the ends of the tube were fixed and covered with gauze, so that the next time he would urinate we would determine whether the bladder leaked into his urethra or into the tube.

[The perineal wound healed per primam. Three days after the catheter was withdrawn the urine, which was kept stained with methylene-blue all the time, showed in the feces. The fistula has not closed, although the frequency of urination is about normal. In other words, his cystitis is better and his prostatic irritation is relieved, but his urethrorectal fistula still exists.—ED.]

LAMINECTOMY FOR BULLET IN LUMBAR SPINE—REMOVAL OF BULLET

HISTORY

Patient, a male, aged twenty-six. November 23, 1912, while hunting, he was struck by a stray bullet, the bullet entering his back on a level with the fourth lumbar vertebra, midway between the spine and the crest of the ilium. He fell over backward into eight feet of snow; was not rendered unconscious, but did not attempt to rise. He was carried to the train and from there to a hospital. The wound was probed that night, but the bullet not removed. Patient could neither flex nor extend his feet and he could not move his toes. The feet felt numb. He could not sit up in bed on account of the pain at the site of the injury. He was catheterized for the first time that night, and regularly for three months following.

At present the patient has incontinence of urine at night and incontinence of the bowels. They are loose. No blood in urine or feces. He can flex his thighs on the abdomen and also flex the legs on the thighs. He can extend both legs, but cannot flex or extend his feet or toes. Feet feel numb; has loss of sensation on plantar surface of feet and on dorsum, but not on inner aspect. Has a dulling of sensation on the outer aspect of his legs as far up as his knees; also has dulling of sensation around anus. Knee and cremasteric reflexes present. Has no girdle sensation; has no sharp pains; no muscle twitching. Had sharp pain in toes two weeks after injury, lasting five days. *He says it felt as though his toe-nails were coming off.* Wound in back healed at end of two months. When attempting to stand, he loses his balance. Past history negative. Venereal infection denied.

EXAMINATION BY DR. C. L. MIX

Reflexes.—The patellar and ankle-jerks were absent, though at first sight the patellar reflexes seemed present, but the resulting motion proved to be an involuntary twitching. The cremasteric reflexes were present and normal. The epigastric and abdominal reflexes were normal to brisk. The plantar reflexes were absent.

Sensation.—Tactile sensation was lost in a saddle-shaped area beginning at the sacrum above and extending around the perineum, down the back of the leg, in the territory of the small and great sciatic nerves to the foot. Expressed in the terms of the innervation of the nerves, there is a preservation of sensation in the anterior crural territory and its internal saphenous branch. The tactile sense is also preserved in the territory of supply of the external cutaneous and genitocrural nerves. The loss of sensation is in the territory of supply of the small and large sciatic nerves, and slightly in the territory of supply of the superior and inferior gluteal nerves. In general, the sensory losses were greater on the right side.

Motion.—All the muscles supplied by the great sciatic nerve were paralyzed. The quadriceps extensors and sartorius, which are supplied by the anterior crural nerves, can be moved reasonably well. The biceps femoris, the semimembranosus, and the semitendinosus muscles are very paretic. All the muscles below the knees and above the anterior and posterior tibial and peroneal groups are very weak. The adductors can be moved to some extent, and so also can the external rotators of the thigh.

Contractures.—The terminal phalanges of the big toes are contracted.

Atrophy.—A beginning atrophy is present, and is confined to the muscles supplied by the great sciatic.

Conclusions.—It is likely that the bullet has lodged at the level of the body of the fourth lumbar vertebra or very near it, causing bone splintering, possibly, or else directly pressing upon the cauda. The long-continued pain which was complained of in the history indicates a long-continued pressure, such as would be exercised either by the bullet itself or by the bone splinters. The lesion is of the peripheral type—a cauda lesion. This is shown by the fact that it follows in general the outline of the nerve-trunks rather than following the supply of the segments of the spinal cord. It must involve the first and second sacral roots and all below, and it probably also involves the fibers in the lumbar region as high as the third. It does not reach the first lumbar root, because the ilio-inguinal and iliohypogastric nerves are not involved. Since the external cutaneous, the genito-crural, and the great bulk of the anterior crural nerves have escaped, the lesion probably involves none of the second or third lumbar roots. The bladder trouble mentioned in the history is due wholly to a lesion of the vesical filaments and not to any lesion of the spinal cord. The cord, which ends at the first lumbar vertebra, is entirely free. I will place the site of the injury as at the fourth lumbar vertebra, and by reason of the unequal distribution of the anesthesia, as being not quite in the center of the cauda, but a little to the right side.

COMMENTS AND OPERATION

DR. MURPHY: What was the date of the accident?

INTERN: November 23, 1912.

DR. MURPHY: What is the matter with him?

INTERN: He has an injury to the cauda.

DR. MURPHY: Where?

INTERN: Between the third and fourth lumbar vertebræ.

DR. MURPHY: What nerve is intact? The anterior crural, is it not?

INTERN: Yes.

DR. MURPHY: Why?

INTERN: Because he is able to flex and extend the thigh.

DR. MURPHY: What other nerve is intact?

INTERN: A portion of the sciatic.

DR. MURPHY: Yes. What nerves are out of commission?

INTERN: The posterior and anterior tibial.

DR. MURPHY: This is not a cord lesion, because the injury is beyond the region of cord segments. It is in the cauda. The bullet rests between the third and fourth lumbar vertebræ. Remember, it is something that involves the cauda. You cannot speak of cord segments here because there are none in the cauda. The central portion of the cauda is injured—just the center; both sides are intact. Those nerves that take their origin from in front are intact; those that come from behind are intact—those that supply the upper portion of the sciatic.

I always like to tell a story about a man who was shot and taken to a police-station. A doctor was called. He came, looked over the man, took off his coat and gloves, and prepared to probe for the bullet. The police sergeant, who was watching, said to the doctor, "I would not probe for the bullet. I have been at the County Hospital, and they say over there that you should not probe for a bullet." The doctor did not continue the probing, and the man was taken to the County Hospital. That was good surgery.

In this case a double mistake was made of probing for the bullet and catheterizing the patient. Never catheterize when the trauma involves the spinal cord. Why? Because the greatest mortality in injuries to the spinal cord comes from bed-sores and infections of the urinary bladder and the kidneys. That is why these patients die. They do not die because the cord is divided. The lesions that cause death are the trophic changes, the bed-sores, and the infections of the urinary tract. Once you start catheterization, the patient is doomed, because infection always takes place. The patient always gets a cystitis, the result of catheterization. No matter how sterile the catheter is, the bladder becomes infected. Just let the bladder fill with urine. After two or three days, if the patient is a woman, massage the sphincter vesicæ through the vagina; if the patient is a man, massage the sphincter through the rectum. Finally, there is relaxation of the sphincter and the urine begins to flow. There is no need to catheterize.

What makes me think that the cord is not cut, but compressed by a foreign body, is that the pain continued in his toes for a long time after the injury. He said he thought his toe-nails were falling off. Pain does not persist in the terminus of a nerve

Fig. 139.—Laminectomy. The bullet is in the midline, between the third and fourth lumbar vertebræ. It is flattened out, and pressed on the cord.

when the axons are cut. Temporarily, the axons are compressed. The fact that he had pain in the toes leads me to believe that his external popliteal axons are intact. They are being irritated, but they are intact. They are not divided.

The management of this case depends on the condition in which we find the axons. If the axons are cut and we can reach them, we will make an end-to-end union. That is the one place in the spinal cord where you can expect regeneration—that is, the caudal zone. The true spinal cord possesses no regenerative power, because in order to get regeneration it is necessary that each axon should have a neurilemma. The neurilemma is the regenerative sheath of the axon. Where the neurilemma is absent, there is no effort at regeneration. The neurilemma is absent in all the nerve-trunks of special sense—the optic, olfactory, gustatory, etc.—and in the true spinal cord. Therefore if there is a division of the spinal cord, no matter if you suture the segments together the instant after the division occurs, regeneration never takes place. Why? Because the cord axons have no neurilemma. In the cauda the axons have a neurilemma, the same as the peripheral nerves. The caudal filaments receive their neurilemma when they leave the commissures of the cord, although they are still within the spinal canal. There is the same power of regeneration that there is in the other peripheral spinal-cord nerves. There is in these structures the same potency of regeneration that there is in bone.

The skiagram shows that the bullet is lodged between the third and fourth lumbar vertebræ, in the middle of the cauda, but a little to the right of the median line. It is flattened out considerably. It must have passed through the vertebræ into the spinal canal and compressed the cord. This is the most difficult place in the whole spinal cord to get to on account of the erector spinæ muscle. It is such a large, strong muscle. Which side did the bullet come in from?

INTERN: Left side.

DR. MURPHY: We will make a longitudinal incision over the spinous processes, placing the third lumbar vertebra in the center of the field. You see the enormous depth to which we have to go here on account of the erector spinæ muscles. The muscles on either side of the spine will be retracted so as to expose the spinous processes. We will remove the spinous processes of the third and fourth lumbar vertebræ with rectangular bone-cutting forceps, and then I will use the rongeur forceps to remove the laminæ. There is the cord shining through. It bulges above the point of injury, but not below. I think I can feel the bullet; at least, I can feel a hard mass between the third and fourth lumbar vertebræ. The resistance seems to be right here. The cord is flattened. Cicatricial tissue has developed here, so that the cerebrospinal fluid will not be lost. I will needle the cord to see if I can determine the exact position of the bullet. The instrument strikes something hard between the third and fourth lumbar vertebræ. It is not the anterior wall of the spinal canal. It may be a fractured bone that has been displaced in that position. The needle passes in only about an eighth of an inch. Higher up it goes in for fully half an inch. Did you learn the size of that bullet?

INTERN: No, doctor. He never found out who shot him or where the bullet came from.

DR. MURPHY: Is one side involved more than the other?

INTERN: No, doctor.

DR. MURPHY: There appears to be normal cord above and below the injury. I will now split the cicatricial tissue in order to remove the bullet. I have made the opening large enough so as to take the bullet out easily. I have split it parallel with the long axis of the axons. There is no escape of spinal fluid because of this cicatricial tissue. The bullet is embedded in the bone, and I have to lever it out. It is very difficult to remove it, not only because of its location in the bone, but because it has flattened out so much and its rough, broken edges fix it in place.

Now it is out. It was right in the middle of the cord. Of course, suturing the dura is out of the question with all that cicatricial tissue present. The best we can hope for here is that the axons have been only compressed, and not severed. They will regenerate if they are not cut. I do not see any cerebrospinal fluid. I assume that I have not opened into the cerebrospinal zone—merely into an adherent cicatricial mass; namely, that the dura and pia have become adherent to the cord in the cicatricial mass. No signs of the escape of cerebrospinal fluid at any time here means that we are free from the danger of cerebrospinal infection. I will close the wound by putting in figure-of-8 silkworm-gut stitches, just as we do in closing the abdominal wall after a laparotomy. We will get all the muscles in apposition over the spinal column, and prevent dead spaces from forming. There was very little bleeding here—none that could not be checked by pressure.

The strength of the spinal column is not impaired at all in these operations. The wound will be dusted with bismuth subiodid and sealed with collodion gauze. A large, plain, sterile gauze dressing will be applied. Later the patient will be advised to wear a leather jacket for a while.

NOTE ON CASE MADE FOUR WEEKS AFTER OPERATION

DR. MURPHY: Let the record show that there is a complete primary union; that the sensation in the limbs has materially improved, and he feels as though his limbs were going to move, while before he felt as though they were not going to move at all, meaning that the muscle sense is returning.

[Muscular action is very greatly improved and is constantly increasing, except the movement of his great and second toes. He has control of the feces and urine.—ED.]

FIXATION OF KNEE WITH BACKWARD LUXATION OF TIBIA

HISTORY

Patient, male, aged thirty-seven, enters hospital on account of limited flexion and extension of left knee. September 14, 1911, he was struck on the knee by a falling mass of earth weighing about 150 pounds. It broke to pieces on striking the leg, and inflicted a small superficial skin cut at the time. He had slight pain in the leg at the time, but the knee was not swollen. He was in bed four or five days, but at no time did he have any pain. September 19th he got up on crutches. After two or three days the knee began to swell and became very painful. He did not have a chill preceding the onset of the swelling and never had any elevation of temperature. He returned to bed, because he could not walk on account of the pain in the knee. The leg was put in a plaster cast for two months, the cast extending from the ankle to the thigh. He had some pain then, but it was not severe. Then a plaster cast was applied, including the whole body. It was left on for three months. When it was removed in February, 1912, the knee was almost stiff. There was slight pain on movement and walking. Seven months ago, September, 1912, the knee was manipulated under anesthesia. After that he noticed that the lower leg was not in alinement with the thigh. It was luxated backward. Motion was not improved. Since that time he has had pain only on continued use, and then only slight. He has a backward luxation at the knee now, with very little motion in the knee; no tenderness. Past history negative; venereal denied.

COMMENTS AND OPERATION

Dr. Murphy: The differential diagnosis as to the pathologic change that has taken place in that knee could not be made from that history. You will see that he has a subluxation backward. That was due to what?

INTERN: He did not notice it until after knee was bent under anesthesia.

DR. MURPHY: How was the leg before that time?

INTERN: It was flexed at an obtuse angle.

DR. MURPHY: That is not the statement he made to me. I do not see how that luxation could be produced in an endeavor to flex the limb. It could be produced in an endeavor to straighten the limb. If you will look at the skiagram, you will see that there is quite a little subluxation of the tibia. There is a fibrous fixation of the patella. There is a porosity of both the femur and the tibia, and there is a little thickening of the femur. There is nothing to be seen in the condyles of the femur, nor in the tuberosities of the tibia, to indicate that it is a lesion like tuberculosis. He has not a complete ankylosis. I made him the proposition that if the lesion was tuberculosis I would do an arthrodesis; that if the lesion was such that an arthroplasty could be performed on the knee, I would do that operation. He distinctly understands that I have the right to decide which one of these procedures I am going to follow. This man has pain in his knee, and that pain will continue until something is done. Nothing is to be gained by delay. The condition will progress until he gets a complete bony ankylosis and that will not improve the situation at all.

On account of the indefinite diagnosis in this case I am going to make a different incision from what I usually make. I am going to make a transverse incision just below the patella, and then I am going to split the patella by means of a double L incision, such as we make when lengthening tendons. Then we can examine the condition of the joint. If we decide to do an arthroplasty, then I will take my flap from the remaining portions of the capsule.

If you will look at the skiagrams made by Dr. Hochrein, showing the blood-supply around the knee-joint (Figs. 135 and 136), you will see how we plan to get a viable skin-flap. The nourishment of this flap, as I take it up in this way, comes entirely from

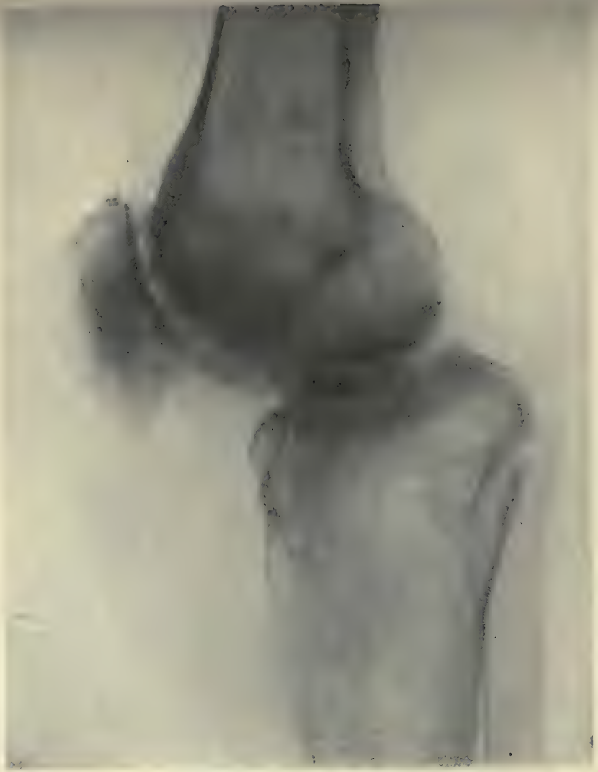

Fig. 140.—Arthrodesis for tubercular knee. Lateral view. Bony fixation of patella to femur and backward luxation of tibia and fibula; slight enlargement of ends of femur and tibia, with porosity of the bone substance. No bony ankylosis between femur and tibia.

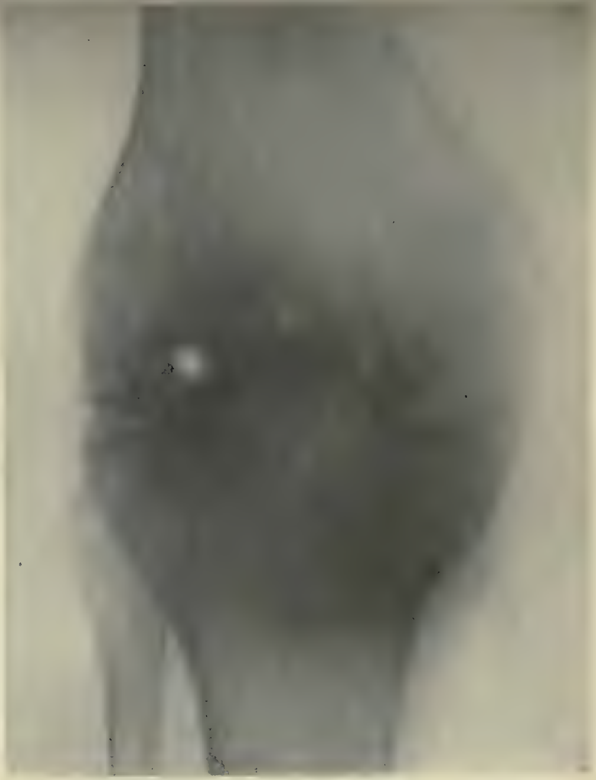

Fig. 141.—Arthrodesis for tubercular knee. Antero-posterior view. The joint cavity is almost entirely obliterated by the thickening of the bones. The joint capsule is destroyed. The interarticular cartilages are thinned out. The enlargement of the articular ends of the femur and tibia and the haziness of the shadows are quite apparent.

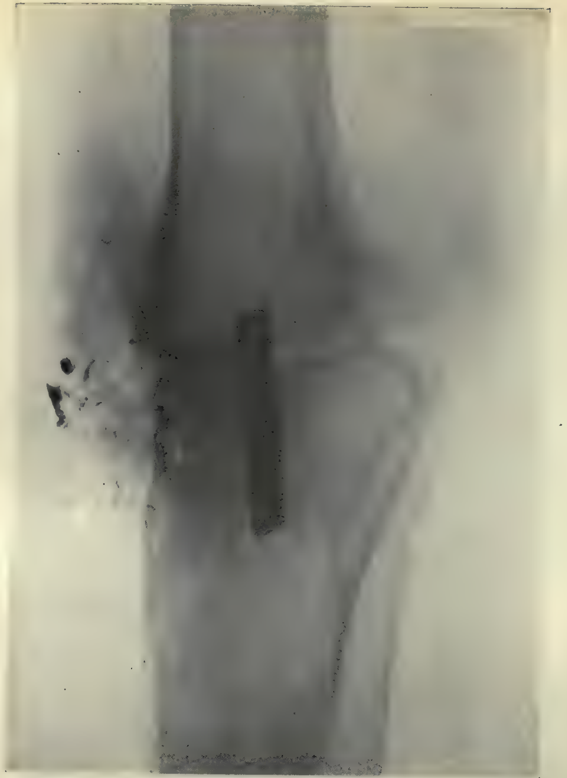

Fig. 142.—Arthrodesis for tubercular knee. Skiagram (lateral view) made ten weeks after the operation of arthrodesis. The leg is perfectly plumb. A plate of phosphor bronze was driven into the femur and tibia to produce immobilization. The patella was removed. Firm bony union.

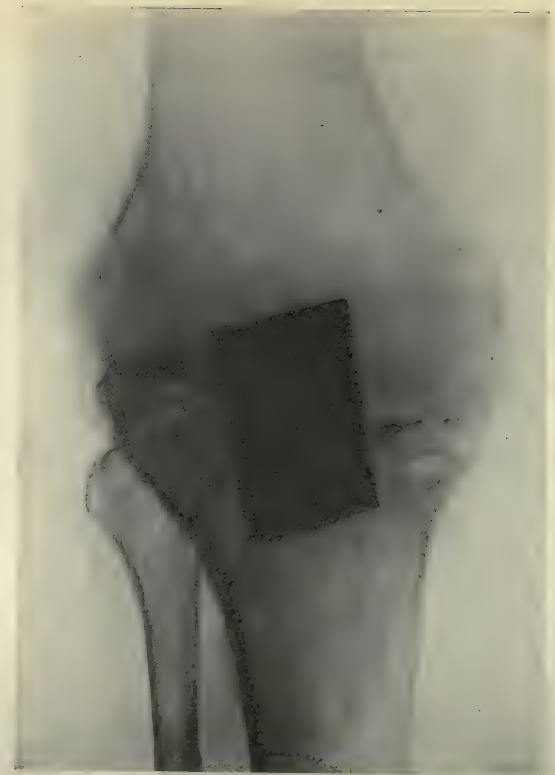

Fig. 143.—Arthrodesis for tubercular knee. Anteroposterior view; skiagram made ten weeks after the operation of arthrodesis, showing the position of the phosphor-bronze plate. Firm bony union between the femur and the tibia.

above. There is a bony ankylosis between the patella and the femur. The patella is firmly fixed. What disease produced that we will endeavor to ascertain as we go farther on with the operation. I believe that the process is tubercular. That means what? That means that we cannot do an arthroplasty. An arthroplasty is then out of the question. We must endeavor to give this man a bony ankylosis of the knee—a fixed knee, in place of a movable knee. With this condition present you can see how futile it would be to put this man under "Murphy's treatment," injecting his knee with formalin and glycerin. Still we have some of these cases come to us very frequently—cases of this class which have received this type of treatment from others. These are not the cases for that treatment. It would absolutely be futile. This is a tuberculosis of the knee, as can be seen from the caseous foci with the mixed infection. We will do a typical resection and not an arthroplasty.

We will remove the patella and this tubercular capsule. What we are going to endeavor to do for this man is to give him a stiff but useful leg. We will endeavor to correct the backward luxation of the tibia. We will square off the ends of the femur and tibia with a Hey saw, removing the articular surfaces of both bones. A slot an inch wide and equally deep will be made in the end of the femur and the tibia with the chisel, transversely, and a phosphor-bronze plate two inches long is inserted into these slots, so as to secure absolute fixation and immobilization. That will correct the subluxation of the tibia and favor bony union.

That plate stays in the one position. I cannot move the leg now either laterally or forward and backward. Having removed the patella, we will unite the ends of the patellar ligament and the quadriceps tendon with chromicized catgut to aid in immobilizing the joint. We will suture these two ligaments together firmly, and the constant traction of the quadriceps tendon will aid in immobilizing that joint during the process of repair.

One of the most important points in treatment in joint repair in these cases of tuberculosis is absolute immobilization. That phosphor-bronze plate prevents motion and immobilizes the joint better than anything else could do it. Its use simplifies the operation very much.

Now we suture the soft or capsular structures around the joint so as to give further mechanic support to the leg. The skin is sutured with horsehair. No drain. The leg will be put in a straight plaster-of-Paris cast. One rarely ever has to use ligatures in this operation. There is little bleeding. This cast will be left on the leg for from three to six weeks, if possible.

[This patient left the hospital ten weeks after the operation with the leg in splendid condition. The skin-wound healed promptly, and firm bony union had taken place between the tibia and femur. He was advised to wear a full-length leather stocking for six months to insure permanency of the result obtained.]

FRACTURE OF THE FEMUR ABOVE CONDYLES, WITH NON-UNION AND OVERRIDING OF THE PATELLA BY THE LOWER END OF THE UPPER FRAGMENT

HISTORY

Patient, male, aged forty-six, comes to hospital on account of limited extension and flexion of left knee with inability to put body weight on leg. Trouble dates from November 30, 1912, when he fell a distance of sixteen feet from a bridge onto the ice. He thinks he landed on the side of his face, left hand, and left knee. He was unconscious for one hour. A diagnosis was made of fracture of the right arm and left leg, with laceration of the skin of the right side. The femur was protruding through the skin above the left knee, and he had a great deal of pain at this point. The wound discharged pus for five weeks and an occasional sequestrum of bone was extruded. He had no chill immediately after the accident, but his temperature was elevated above normal all the time. Ten days after the accident he had a severe chill, followed by a high temperature. He says he had pneumonia. His temperature was 105° F. Four weeks after the accident, while the wound was still discharging, the leg was put into a plaster-of-Paris cast. The wound discharged for one week more. The cast was removed at the end of four weeks. Since that time he has been unable to bear his body weight on the leg. All the anatomic landmarks around the knee are absent on account of the infiltration. Motion in the joint is limited. The external condyle of the femur is prominent. Past history: Venereal denied. Family history negative. No chronic cough, night-sweats, or evening rise of temperature.

The skiagram shows a non-union of the fragments with the overriding of the lower fragment anterior to the patella and the knee-joint. The fracture is almost transverse, about two inches above the condyles.

Dr. Murphy (Operation April 29th): This is not a pleasant case to handle. He has had a fracture of the lower end of the femur, about four inches above the articular surface of the bone. The lower end of the upper fragment has passed in front of the lower fragment, and overlaps it about two inches. There is no union, although much callus has formed at the ends of both fragments. I am going to see if I can take off enough bone from the upper fragment to bring it back into place, and then try and secure some sort of apposition of the fragments. I want to save the joint if it is possible, although the patient is satisfied to have an ankylosis of the knee. He cannot go on in his present condition because he has a non-union. His leg is not only useless, but it is painful. He seems to have a fibrous union there, but that is useless.

We will make a curved incision, convexity downward, across the front of the thigh over the seat of the fracture and retract the skin-flap upward. By drawing the quadriceps to one side, the fracture is exposed.

The line of adhesion between these two fragments is plainly to be seen. I believe that in spite of this dreadful deformity I can give this man a serviceable leg, and, I hope, with a movable knee-joint. I will not do a transplantation operation, but will implant two phosphor-bronze plates at right angles to each other, on the same principle we follow in an arthrodesis. That procedure will entail less trauma to the tissues, and in this case will lessen the chance of infection, thus materially favoring a successful outcome. Remember that the essential point in all this work is to avoid hand contact with the wound, and not to drag anything over the skin into the wound, even though the skin has been disinfected thoroughly with iodin. There is no constrictor on the leg, still there is little bleeding. By putting in two metal plates at right angles to each other the fragments cannot move backward or forward or laterally. The plates also maintain perfect apposition, the contraction of the muscles aid-

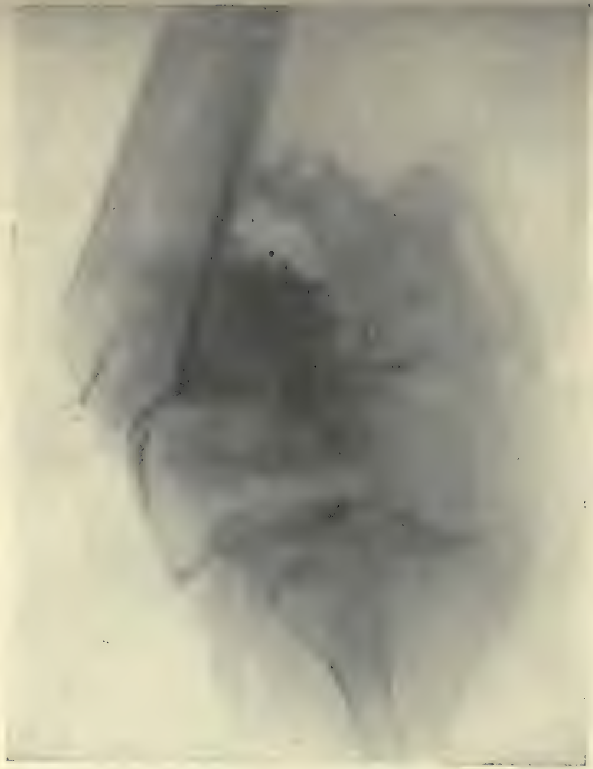

Fig. 144.—Ununited fracture of the femur. Lateral view, showing fracture of femur above the condyles, with displacement of shaft forward and downward in front of the lower fragment and to the outer side of the patella. The lower fragment is rotated forward. No union.

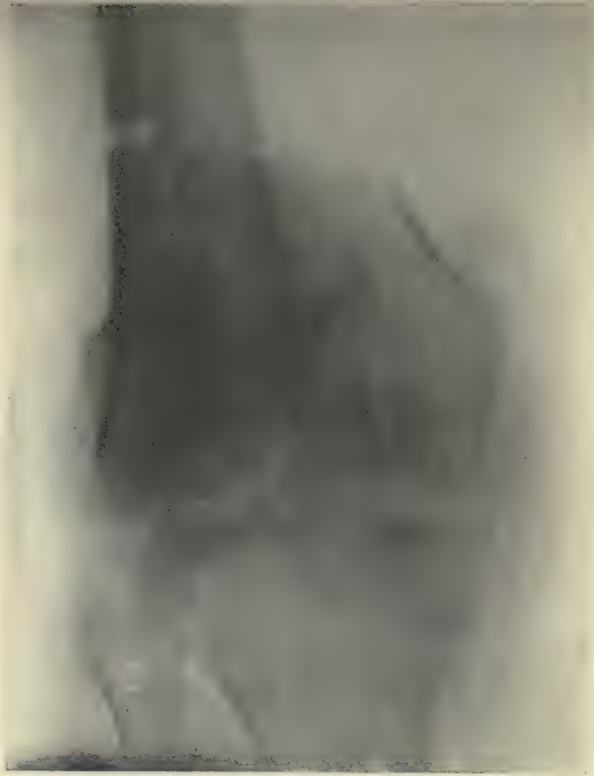

Fig. 145.—Ununited fracture of the femur. Antero-posterior view, showing the relative position of the upper fragment to the condyles and the patella. The forward rotation of the lower fragment is not shown in this picture.

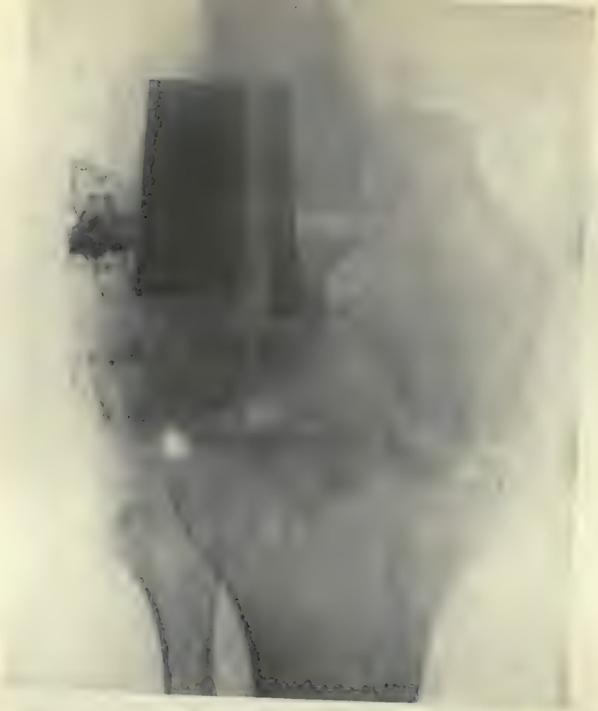

Fig. 146.—Ununited fracture of the femur. Antero-posterior view made three weeks after the operation. After one and three-fourth inches of the upper fragment had been cut off and the ends squared, two phosphor-bronze plates were inserted into both fragments at right angles to each other, thus preventing motion of either fragment in any direction and securing absolute fixation and immobilization. There was motion in the joint at this time. The phosphor-bronze wire sutures to the left of the plate were inserted into the deeper layers of the wound.

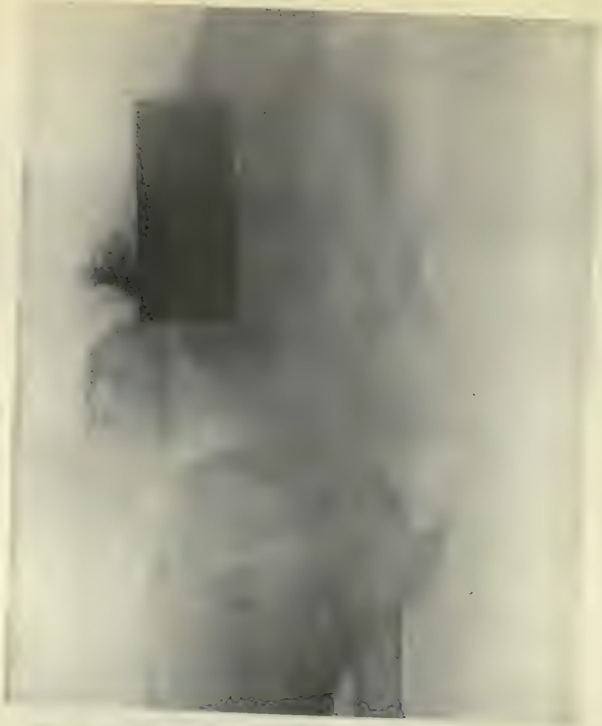

Fig. 147.—Ununited fracture of the femur. Lateral view, made three weeks after the operaton. The leg is plumb. One of the phosphor-bronze plates shows as a thin black line to the right of the broad plate. These two plates were inserted at right angles to each other.

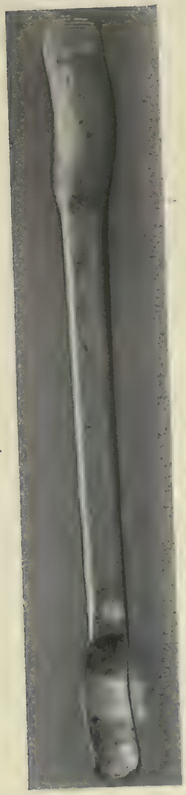

Fig. 148.—Dr. John B. Murphy's bone skid, used to replace luxated bone fragments in the treatment of ununited fractures and to replace the dislocated head of the femur in the acetabulum.

ing by forcing the fragments together. Whatever result he gets, it ought to be either superlatively good or nothing.

We will partially divide the quadriceps tendon so as to have a better exposure and a more accessible field to work in. We will first free the lower end of the upper fragment from its attachment to the anterior surface of the lower fragment by dissection, and then similarly free the upper end of the lower fragment. Next we will remove 1¾ inches from the tip of the upper fragment with a Hey saw, and square off the end of the lower fragment with bone-cutting forceps. That permits us to make the reduction. The two fragments easily come into perfect apposition and alinement. With the aid of our bone skid this is easily accomplished.

Now we will insert the two phosphor-bronze plates at right angles to each other and removed from each other one-fourth of the circumference of the bone. Two slots are made in each fragment with the small buzz-saw, and then the plates are inserted, first into the upper fragment and then, by making traction on the leg and straightening the whole limb, the plates slide easily into the slots in the lower fragment. The ends of the fragments are held firmly together and the limb is in a perfectly straight position. The whole procedure was accomplished with a minimum amount of manipulation and with less trauma to the neighboring structures than one would imagine was possible in a case like this, especially considering what was finally accomplished in the way of securing accurate apposition and reduction.

The skid we used is a great help in the work. It gives just the help needed to swing a bone into any desired position. We were not inside the joint at any time during the operation.

We will suture the ends of the quadriceps tendon together with chromicized catgut and close the deeper layers of the wound with plain catgut. The edges of the skin incision will be approximated with horsehair, the iodin removed with alcohol, and the wound dusted with bismuth subiodid and sealed with collodion. No drain.

Let the record show that the fracture was exposed by means of a curved incision on the front of the thigh, and the ends freed with the periosteotome; we squared off the end of the upper fragment with the saw, and of the lower fragment with bone-cutting forceps. Two phosphor-bronze plates were inserted in the fragments at right angles to each other, to maintain apposition and immobilization. The wound was then closed.

We will just put that leg in a straight plaster-of-Paris cast now, cutting it with the Gigli saw as soon as it has hardened, as is our custom. That will permit the cast to spread if there is any swelling of the leg. The first dressing will not be made for three or four weeks unless untoward symptoms arise. But we have succeeded in doing this operation under the strictest aseptic precautions, so that we do not fear infection. I feel that he will have a limb that he can use.

[The patient left the hospital after nine weeks with good union in his leg, and he had about 10 degrees of voluntary motion in his knee. This will surely increase to 90 degrees. He was up and around on crutches, but was advised to wear a cast for six months.]

CYLINDRIC-CELL CARCINOMA OF THE BREAST

HISTORY

Patient, female, aged fifty-three, enters hospital on account of a hard, indurated mass of the size of a pigeon's egg in her left breast, one inch above and to the outside of the nipple.

Patient first noticed the mass in her breast in February, 1913. It was then hard, and of the same size as it is now. She thinks that she had a small mass the size of a pea in the breast three years ago, but is not positive. She remembers injuring the breast quite severely two years ago. In December, 1912, her heart and lungs were examined thoroughly, and no mass was noted at that time. The mass is not adherent to the chest-wall, and the skin over the mass is movable. There is no ulceration present, no retraction of the nipple, no axillary lymph-node enlargement. The breast is not painful, but is slightly tender. She has six living children; the last was born ten years ago. After the birth of the third child she had puerperal sepsis and milk-leg. Menopause at forty-three.

Patient had jaundice in 1900. Mother contracted pulmonary tuberculosis when patient was eighteen months old, and died four and one-half years later. Father died of pulmonary tuberculosis when patient was four years old. Paternal grandmother died of carcinoma of the left breast.

[For a full description of Dr. Murphy's technic of the operation for cancer of the breast see the December, 1912, Clinics, p. 779.—Ed.]

COMMENTS AND OPERATION

Dr. Murphy (Operation, April 29th): It does not seem possible that a physician examining her chest four months ago could have overlooked the tumor, and still it does not seem possible that the tumor could have grown to this size in so short a period as four months. Besides, it would scarcely grow to that size in so short a time. The tumor is movable on the chest-wall; the skin over it is likewise movable. It is firm, nodular in character, and in no wise an infiltrating growth. There is no retraction of the nipple. We will excise this mass and examine it microscopically (frozen section) before we decide definitely whether it is necessary to remove the entire breast and clean out the axilla. We will encircle the tumor by making two elliptic incisions, and extending down to the pectoral fascia. Judging by the solidity of this tumor one would say that it is not a cyst, but one cannot differentiate between a cyst and a solid neoplasm. It is more likely to be an adenoma or a fibro-adenoma. Aspiration of the mass before operation was negative. I am not going to take out the breast unless this proves to be a malignant neoplasm.

It is readily dissected free from the surrounding tissues. We will close the incision temporarily with fixation forceps while we await the report of the pathologist.

On incising the mass it is seen to be a solid tumor. It looks like a fibro-adenoma, rather than a carcinoma.

[Report was made by the pathologist a few minutes later that the mass was a cylindric-cell carcinoma.] The report of the pathologist makes it necessary to do a radical operation in this case.

We will encircle the breast by the usual Halsted incision, extending out on to the shoulder. The breast will not be removed until we have cleaned out the axilla. First we remove the anterior aponeurosis of the pectorals and all the fatty tissue. Then the pectoralis major is freed from its costal attachment and reflected outward onto the arm, splitting it well up to the clavicle. The fascia and fat between the pectorales major and minor are then removed, as a lymph-node is commonly found in this location. The pectoralis minor is then detached from its costal attachment and is deflected outward, or it is removed entirely. You must remove all the aponeuroses of both these muscles. Dissect free the edges of each muscle with dissecting scissors, pass two fingers under the muscles, and divide with the scissors close to the ribs so as to leave enough muscle distally to fill in the axilla; that is, to make an axillary muscle-pad. The chest portion of the muscles is elevated entirely, so as to expose the fascial covering of the ribs. There are no lymph-structures here in the muscle. Now we are ready to dissect the axilla. We will take out all the lymph-nodes and lymph-carrying tissues before we manipulate the breast at all. By going at that from above you lessen the chances of metastases from the breast into the general lymph-system and you are always master of the whole situation. The axillary artery and vein are freed very carefully of fat. There is very little hemorrhage here. Note that we always carry out dissection from above downward. We have cleaned out the axilla thoroughly; there is the clavicle. Remember the subscapular nerves. If I cut them, she could not put her arm behind her body to fasten her skirts, as the latissimus dorsi would be paralyzed.

Now I am going to remove the breast itself. There are the pectoral muscles. We will deal with them as already mentioned. Occasionally there is a lymph-node in the fat on the muscle between the two pectorals. In removing the breast we always undermine the skin-edges as far as possible. That is a very important step in the radical operation for breast cancer.

This woman stated that there was no tumor in the breast in December (four months ago), when an examination was made of her heart and lungs. In making an examination of the lungs and heart, the breast is not always examined, so that must not be interpreted as meaning that she did not have the tumor at that time. The doctor was not looking for it. Unless a man is examining the breast deliberately, he may not detect a tumor in it.

There was no apparent enlargement of the axillary lymph-nodes in this case. The subsequent contraction of a scar might compress the axillary vein and cause edema of the arm. Greenough found it in 30 per cent. of cases. That is the purpose of leaving these muscle supports—these pectoral muscle-stumps—to fill in the axilla and act as a protecting pad for the axillary vessels and nerves. If you do not do that, what must fill in the axilla?

Cicatricial tissue. Nothing else can fill it in. We are very careful to check all bleeding points by ligation.

The next step is to place the pectoral muscle-stumps into the axilla. By means of a few catgut sutures they are swung inward, downward, and backward into the axilla, and fastened to the edge of the latissimus dorsi and the lateral chest-wall. Now we have all the axillary vessels and nerves fully protected. She can swing her arm in any position without interfering with the circulation in the arm. We close the wound by putting in a considerable number of silkworm-gut tension sutures and horse-hair for the edges of the incision. There must be no tension on these flaps. It is necessary to drain every case like this and allow the primary wound secretion and blood to escape. We put in a small rubber drainage-tube extending high up into the axillary space, and fix it to the skin with a silkworm-gut suture. The drainage-tube will be taken out at the end of seventy-two hours. The arm will be dressed by the side of the body, the hand resting on the chest. The iodin is first removed with alcohol, and then the bismuth subiodid dusting-powder is applied. The wound is not sealed with collodion.

Let the record show that this was a scirrhus carcinoma as was determined by microscopic examination; that we did a typical amputation of the breast, cleaned out the axilla, and sutured the pectoral muscle-stumps into the axilla according to our usual procedure.

Now as to the lines of metastases. The first place is through the axilla; the next is in the second interspace, close to the sternum; the third route is the sixth interspace, in the anterior axillary line; the fourth position is the supraclavicular fossa; the fifth route is Handley's, by the chain of lymphatics to the umbilicus and into the abdomen. Handley's scheme of operation consists in taking off all the anterior abdominal wall in cases of carcinoma of the breast. Still another route of infection is from the skin, in a radiating manner, from the zone of primary infection.

I always like to study the gross specimen myself, because often one can make as positive a diagnosis from such an examination as with the microscope, and sometimes a more accurate prognosis can be made. There are many adenocarcinomata that are on the border-line between malignancy and benignancy. If you examine the gross specimen, you can usually tell from the appearance whether it is a scirrhus carcinoma. This is a cylindric-cell carcinoma and that corresponds exactly with the history. She is a sparely built woman, and is very much safer against recurrence with that small amount of connective tissue than would be a fat woman of the same age. Remember, with this amount of connective tissue these tumor cells are easily encapsulated and held in loco. In many of these cases metastases do not form. I do not recall a single case of carcinoma in a woman between forty and forty-five, stout or fat, that did not eventually die a cancer death, and practically all from distal metastases. They rarely return in loco, but in distant organs or tissues—liver, lung, bone, etc.

We did not operate too early. Remember, we do not know how early the malignant cells are carried through the lymphatics. They may go through the lymphatics rapidly, if they go through like the streptococcic infections, or slowly, like the staphylococci. If they metastasize, they metastasize early. That is what happens in fat women, as a rule.

[The drain was removed on the fourth day. There had been little drainage. Stitches removed on the fifteenth day. Primary healing. The convalescence was uneventful. Patient left the hospital five weeks after the operation, having had *x*-ray treatments in the mean time.]

TUMOR OF THE RADIUS

HISTORY

Patient, female, aged twenty-eight, single, enters hospital on account of swelling, tenderness, and pain over the lower end of the right radius, just above the wrist, on its posterior surface.

Early in September, 1911, the patient noticed a slight enlargement and had a dull pain in the lower end of the right radius. She says that she injured the arm very slightly at this point during July. The pain and swelling gradually increased in severity and size. Pain became more intense with use of the wrist. Had no chills or fever. Swelling gradually increased in size until September, 1912. At that time a diagnosis of tuberculosis of the bone was made, and the lower end of the radius, which was enlarged to the size of a pigeon's egg, was opened, cureted out, and the cavity in the bone filled with bismuth paste. Primary union in two weeks. Pain still continued, but did not become severe enough to annoy her until the first of April, 1913, when it became very severe, and the mass began to enlarge gradually and became very tender. She still had no chill or fever. At present (April 29, 1913) the mass is the size of a pigeon's egg, hard and tender to touch. Personal history otherwise negative. Family history negative to tuberculosis and carcinoma.

COMMENTS AND OPERATION

Dr. Murphy: Whether this was a case of osteitis fibrosis cysticus I do not know, because we did not do the original operation. The mass is increasing in size because of the irritation that is being caused by the bismuth paste. This is one of the cases for which the bismuth paste was not intended by Dr. Beck. It was probably a bone cyst originally. It was opened and cureted and filled with that non-absorbable material. There is no evidence there that the bone itself is increasing in size at its periphery. The neighboring tissues are not infiltrated. The radius is crowding the ulna over and is causing a luxation of the ulna from its articulation with the carpal bones. The arm is more painful than it was. We are going to open this cavity, clean it out, and then decide what to do. It was diagnosed recently as being nonmalignant. There is a fluctuating area just in front of the mass that is just a little suspicious of sarcoma.

We are going to make our incision on the posterior surface of the arm, just over the forearm, on the same line as the previous incision. As soon as the knife passed through the skin the bismuth paste was evacuated. That was a fluctuating mass. I could feel it yesterday fluctuating outside the bone. It was the bismuth paste which had escaped from the cavity in the bone. Beck is not to be charged with this result. The paste is not intended to be used in cases such as this.

We will remove all this paste and curet the walls of this cyst thoroughly. I want to see whether this débris is malignant or whether it is simply granulation tissue, the result of the irritation caused by the paste. As I look at this bone it does not appear to be a malignant lesion. It looks as though the irritation came from the foreign material. The bone is hard and firm, not spongy at all. That granular material looks like the lining of the sac—not a suppuration sac, but an irritation sac. I will take out all that granulation tissue. My interpretation of this case is that it is not malignant at all. It is merely a bone cyst, and, therefore, bone should be transplanted. I am going to loosen the periosteum from the walls of the cyst just around the edges, so that I can fracture this bone and throw it into the cavity in place of taking a fragment of bone from her tibia, which I had intended to do before I knew what the condition present was. This is done very much like the dentist prepares a tooth for filling. That bone is just as good as a transplant taken from the tibia. I merely break down the edges of the cyst with a chisel and drop the bone fragments down into this cavity in the bone. The periosteum is allowed to close in over the cavity.

Let the record show that the bismuth paste had escaped from

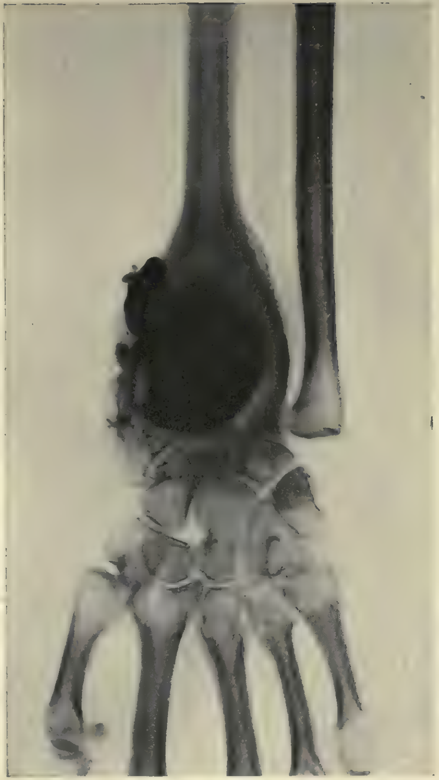

Fig. 149.—Cyst of lower end of radius. Anteroposterior view. The end of the radius is very much enlarged by the cystic formation. The cyst is filled with bismuth paste. The radiocarpal articular surface is broader than normal.

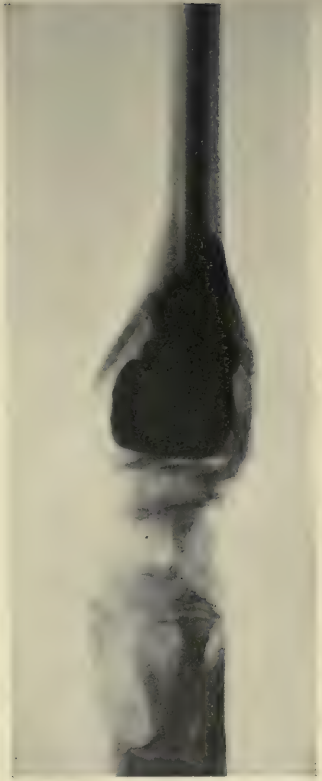

Fig. 150.—Cyst of the radius. Lateral view, showing the enormous thickening of the radius and the cyst filled with bismuth.

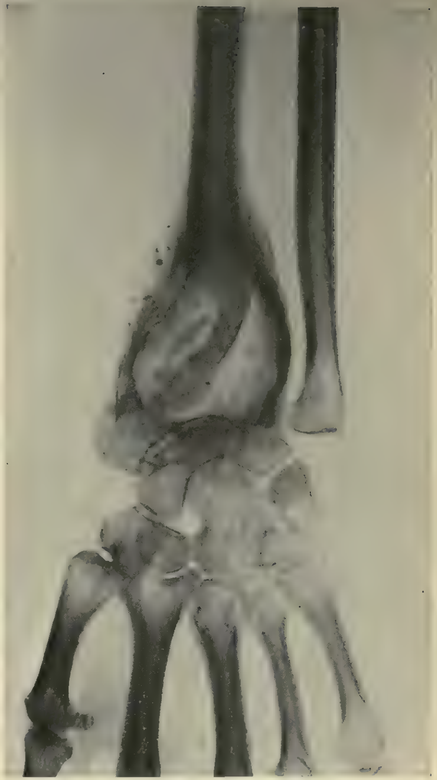

Fig. 151.—Cyst of radius. Skiagram made three weeks after the operation. The bismuth was removed from the cyst, the walls were thoroughly cureted, and pieces of bone were chiseled off the lateral edges and dropped into the cyst to aid osteogenesis.

the bone and was resting under the tissue of the forearm, which accounted for the fluctuation. After removing the bismuth the granulation tissue lining of the cavity in the bone was removed with the curet; then the canopy of the bone cavity was fractured and luxated into the cavity, to aid in the ossification and repair, in place of putting in a piece of the tibia. There is sufficient bone here to fill the cavity and to insure the production of ossification. The lower layers of the wound will be closed with catgut and the skin with horsehair. A sterile dressing will be applied, and the arm will be carried in a sling. It needs nothing else.

[The patient left the hospital after three weeks. The wound had healed and the patient was free from pain and discomfort. The skiagram shows the fragment in the bone cavity.]

ANKYLOSIS OF KNEE-JOINT, WITH HYPEREXTENSION OF LEG AND EXCESSIVE PRODUCTION OF BONE SUBPERIOSTEALLY—ACUTE INFECTIONS IN 'JOINTS; FORMALIN-GLYCERIN TREATMENT

HISTORY

Patient, male, aged thirty-eight, entered hospital April 22, 1913, on account of stiffness and very limited motion in the right knee. Toward the latter part of October, 1910, he contracted a Neisserian infection. Five or six days later the discharge began and continued for ten days. He does not know the exact date in November in which the discharge began. About December first he began to have pain, dull and aching in character, in the right knee. Does not remember having had a chill. On the third day the pain became very severe and the knee began to swell. He had some elevation of temperature at this time, but does not know how much. The knee remained painful and swollen, with slight elevation of temperature, for two and one-half months. No cast or extension was applied. When the pain and swelling subsided, the knee was manipulated under anesthesia twice a week for eight months. The patient was in bed all the time. At present there is practically no motion in the knee. It is painful on continued use. Patient had no sore throat, cold, or carbuncles at time of onset of the trouble. Past history: Diphtheria at seven years. Tracheotomy done at that time. Family history negative.

COMMENTS AND OPERATION

Dr. Murphy: The only history of infection in this case was the Neisserian infection, which he contracted the latter part of October, 1910. The discharge appeared early in November, continued for ten days, and the metastatic arthritis was manifested about the first of December, about eighteen days after the discharge ceased. That is the typic course of a Neisserian arthritis. It practically always appears in from eighteen to twenty-two days after the urethral discharge appears.

With a continuous infection of the Neisserian type traumatisms under anesthesia, such as forced flexion and extension, are very detrimental to the subsequent usefulness of the joint—in fact, they favor the occurrence of ankylosis rather than prevent it. These traumas often cause hemorrhage into the joint, as can be demonstrated by aspiration. This favors the formation of connective tissue within the joint.

An examination of the skiagram shows that this is a fibrous and not an osseous ankylosis between the femur and the tibia and between the femur and the patella. It appears to me that some time during the manipulations of this joint under anesthesia the ligamentum patellæ was torn loose from the patella, because there is some thickening of the lower outer edge of the patella. The leg is hyperextended; in fact, there is a backward luxation of the tibia. That is probably the result of the frequent manipulations of the knee.

The history, as you see, is a typic one. It is the regular course of events in this type of metastatic arthritides, and I wish that every one present would become impressed with the fact that in cases of infection in a joint we must not trust to Providence to effect a cure. We must do something and do it right promptly—NOW! Do it with a purpose.

Acute infections in joints should be considered as one would an acute infection in the soft parts. Prevent the destruction of tissue, first, by *aspirating* the fluid in the joint, second, neutralizing the products of infection by injecting a 2 per cent. solution of formalin in glycerin (at least twenty-four hours old); third, relieve pressure on the ends of the bone by putting on a Buck's extension. In cases of acute infection in any of the large joints *never* put on a plaster-of-Paris cast. In cases of tubercular infection in any of the large joints *always* put on a cast. Our fathers were right in their treatment of chronic joint infections.

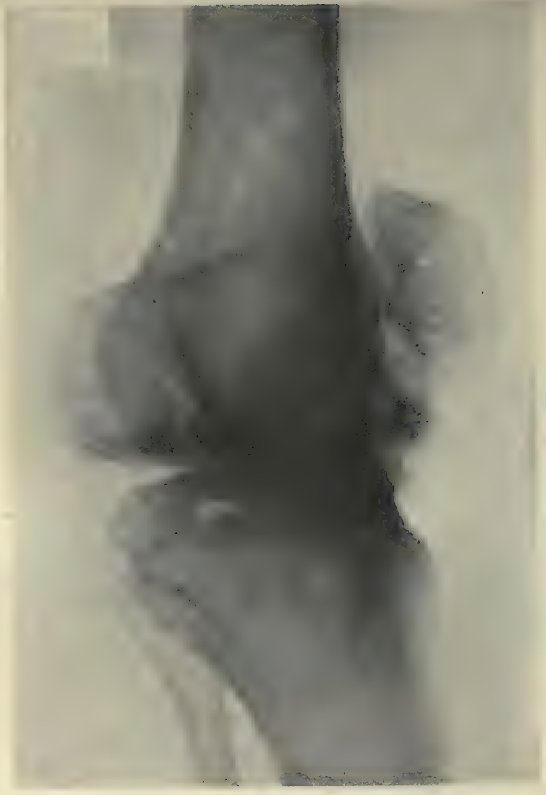

Fig. 152.—Ankylosis of knee. Lateral view taken before operation. Hyperextension of leg with backward luxation of tibia. Excessive production of bone beneath periosteum. Old rupture of patellar ligament. Not a bony ankylosis.

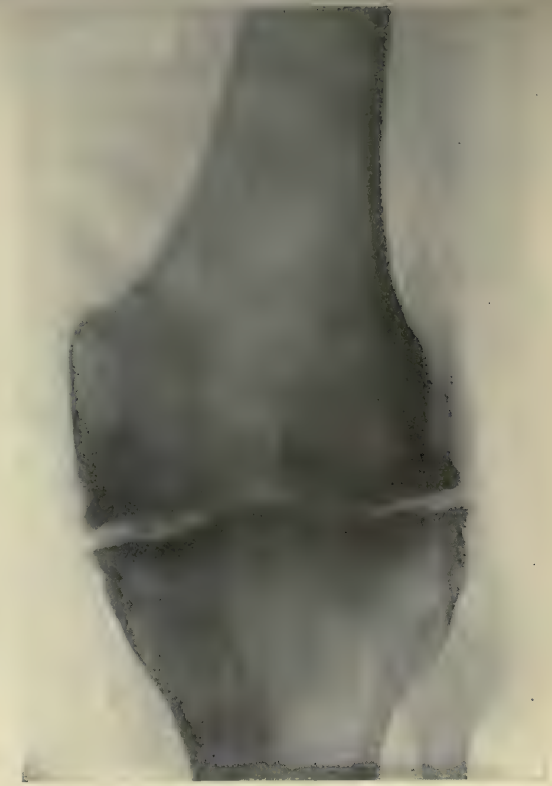

Fig. 153.—Ankylosis of knee. Anteroposterior view before operation, showing thickening of the articular surfaces of the femur and tibia. Exostoses on the edges of both bones. Flexion was limited to 20 degrees.

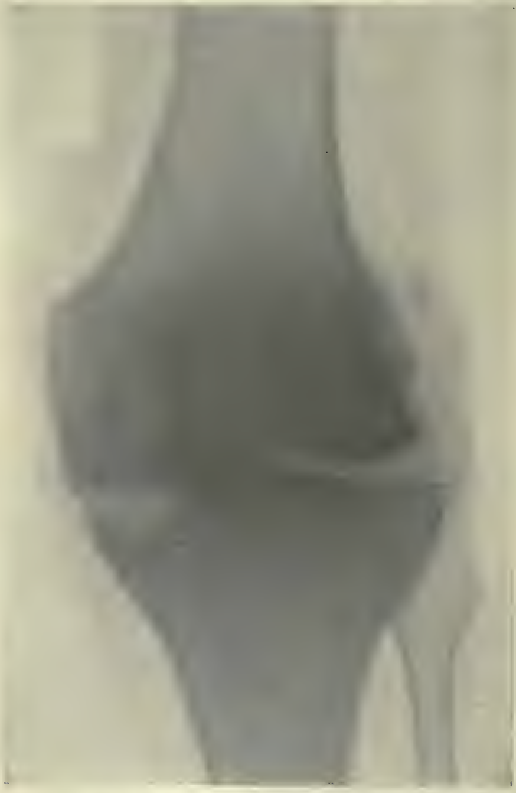

Fig. 154.—Ankylosis of the knee. Anteroposterior view made six weeks after operation. The position of the bones is good. The joint cavity is of practically normal size. Patient had motion in the joint.

They always treated a tubercular joint infection by immobilization. To do that in a case of acute infection is disastrous to the joint. It will surely be destroyed and ankylosis result if the infection is of severe grade. That is the essential point to be remembered. Always treat an acute infection in a joint first by putting on a Buck's extension; second, by aspirating the joint. After you have aspirated the joint, inject the formalin and glycerin solution, 15 c.c. in the case of the knee. We insist that the solution should be mixed at least twenty-four hours before using. Never use a solution that has not been mixed that long. Why? Because it takes sixteen hours for formalin to dissolve in glycerin. If you will look at it through the microscope in twelve or thirteen hours, you will see little globules of formalin in the glycerin. After sixteen hours, then, the formalin will be dissolved fully. We wait twenty-four hours to be absolutely on the safe side. Inject 5, 10, 15, or 20 c.c. of the solution, depending on the size of the joint.

What does that do? It excites a chemic inflammation and a coffer-damming of the lymph-spaces in the joints. Formalin when injected into a tissue produces the greatest degree of polynuclear leukocytosis, more than any other material, except Venice turpentine. Remember that the tuberculous process, different from an acute septic process, is not cured by the aid of the polynuclear leukocytes. It heals by granulation, with the formation of connective tissue and encapsulation, while the acute infection is combated successfully by the polynuclear leukocytes. They die and yield trypsin, which converts the albumens into soluble peptones. In the case of old, suppurating cavities, if you inject a material that stimulates polynuclear leukocytosis, you favor resolution and healing.

Supposing we had this case of Neisserian infection from the beginning, how would we have conducted the treatment? Immediately after the chill or the manifestation of the infection in the joint we would put on a 25-pound Buck's extension; then we would make an autogenous vaccine from the urethral discharge and inject it in sufficient quantity and often enough to produce the desired effect. Next, we would have aspirated the excessive fluid in the joint and inject the knee-joint through the same needle with 15 c.c. of a 2 per cent. solution of formalin and glycerin. If the temperature remained elevated materially above normal,—100°–100.5° F. or more,—showing a continued absorption of the products of infection in the joint, we would aspirate it again in twenty-four hours, and again in forty-eight hours, probably without injecting it again, unless the temperature was very high. Just take off the tension by aspiration, but do not repeat the formalin and glycerin injection until after forty-eight hours. If the temperature is still above normal, then aspirate *and reinject.* You rarely need to make more than two injections of formalin and glycerin. If necessary, aspirate again in seventy-two hours, but do not do any further injecting. In three and one-half weeks that knee would be perfectly well. You would have cured the patient. Remember, that if you do not separate the joint surfaces by applying a Buck's extension, you favor the occurrence of an ankylosis as a result of the destruction of the soft tissues in the joint by the intra-articular tension, due to voluntary muscular contraction the result of the presence of the infection in the joint. This type of infection is always ruinous to the joints unless it is treated properly and early. Remember, this treatment has no value once the joint is destroyed and the ankylosis has occurred. Never drain these joints, because that admits additional infective microörganisms into the joint from without and the subsequent destruction of the joint is inevitable.

By examining the skiagram you will note that this man has a hyperextension, just the opposite to what usually occurs. There is an excessive development of bone in front. The patella is fixed on one side only. We must take the greatest amount of bone off from behind. We must also elevate the patella. We are not going to be able to rotate the patella (turn it turtle) because we cannot take off enough bone to permit of this procedure without making a flail-joint.

We do not put a constrictor on this leg. The hemorrhage in these cases is not excessive, and we prefer to ligate bleeding points before closing the wound at the end of the operation. If you will look at the circulation of the knee as shown in the skiagrams (Figs.

135 and 136), you will see the source of the nutrition for the skin and fascia flap.

We will make the usual incisions, one on either side of the patella. Each incision is about six inches in length, curving backward above so as to make a curvilinear incision, the convexity directed toward the patella. The upper extremities of the incisions are four inches apart, the lower, three inches.

I am going to try to secure all my interposing flap from one side, instead of two sides, as is our custom. Having dissected the two skin and fascia flaps free, I will carefully dissect the interposing flap or flaps, if I need two. Remember, each flap consists of fat and the fibrous layer of the capsule. It is tongue-shaped, the base being downward over the internal and external surfaces of the upper extremity of the tibia. I have secured a very good internal flap, but I will have to make an external flap as well, to slip in under the patella. I have to take off some of the upper extremity of the tibia in order to get the flap in place. I do that with the chisel—a large curved carpenter's chisel. The patella is quite loose—loose as far as the bone is concerned, but not loose as far as the fibrous tissue is concerned.

Now I am preparing the external flap, taking off the skin and leaving some of the fascia and fat. I am just in front of the external popliteal nerve, but I will not include the fibrous portion of the joint-capsule in this flap. I am going to use this as my subpatellar flap. I will have to treat the patella differently from what I do in the average case. One reason why we do not use a constrictor in these cases is that we prefer to have the bleeding take place now, otherwise a hematoma is produced later, after the wound is closed, and that is a serious drawback to the final result. We have had several cases of spontaneous hematoma, and we are profiting by our experience. Having divided the capsule on the external surface of the joint, we will remove as much of the upper part of the tibia as is necessary to give us a straight limb and to relieve the tension on the quadriceps tendon and the ligamentum patellæ and to prevent pressure on the interposing flap. We reproduce the normal anatomic conformation of this bone as nearly as we possibly can, so that the leg will be plumb and joint function normal. That patella appears to me to have been broken. If you could look in here you would see a greatly deformed joint, with the synovial membrane entirely changed, thick, fibrous, and roughened on its surface. That joint appeared to have quite a little motion in it before I started the operation, but you will remember he had a hyperextension of the leg. You can see now that that is due to an enlargement backward and downward of the condyles. We have freed the patella from its attachment, and by retracting it to the side we get a good view of the joint. The hyperextension in this case makes it different from the average case of ankylosis in which there is usually flexion of the limb, varying in degree with the pathologic conditions present.

That is a mighty hard field to work in with a short patellar ligament. There is an osteoma, a production of new bone, between the two condyles of the femur behind. I think you can see that bone. You can see why I am careful in using the chisel there. I am right above and in close proximity to the popliteal vessels. Because of the hardness of the bone, the chisel does not cut as well as usual. However, we are restoring the normal conformation of the tibia and also of the lower extremity of the femur, and we are now ready to interpose the flaps. We will examine the patella. Elevating it, I can see that there was a fibrous union with the femur. I want you to see it—a firm, fibrous union of the patella. I am going to free the quadriceps tendon for some distance up so as to let the patella come perfectly free. Now I find that I must round off this femur in front. In place of being round it is almost squared off so as to make a sharp angle. We can do nothing with that unless we round it off. I will elevate this fibrous tissue from the lower end of the femur before taking off the bone, and I will use that tissue as a part of the interposing flap.

Now we are ready for the interposition of our flaps. You will note how I have increased the depth of the intercondyloid notch and rounded off its edges so as to let the patella slide freely up and down on the femur.

This case gives you some idea what the Neisserian infection does when it lodges under the periosteum and produces this excessive mass of bone. It is practically an osteoma and in this case it was of large proportion. It is a subperiosteal growth of bone, diffuse in character.

Now I will draw the interposing flap from the internal side into the joint. It covers the entire surface of the tibia. We will fix it to the edges of the tibia in front and on the internal side with a few chromicized catgut sutures and also to the stump of the posterior crucial ligament and the posterior layer of the capsule. That covers the space between these bones with fibrous tissue and with fat. This will not absorb. You can see how large and thick that flap is and how much of it we have. We have flap to spare. The smaller flap is slid in under the patella and sutured to the periosteum of the femur. That worked out better than I thought it would. It is the only way I could figure out permanently to mobilize the patella in this particular case. That will effectively prevent the occurrence of a bony ankylosis, and this man should get a good joint here. All raw bony surfaces are covered with fat and fascia flaps. There is no tension on them. The leg is plumb. The enormous quantity of bone produced here in the form of a subperiosteal osteophyte, due to the irritation of the infection, made this a very difficult case to handle. It caused the hyperextension of the leg that was present; it did not show in the skiagram.

Let the record show that we exposed the joint in the usual manner by means of two incisions; that after the bone was removed a flap was made from the inner side that passed clear across between the tibia and the femur, and a smaller flap was made from the outer side that passed between the patella and the femur, so that we had two separate flaps in order to secure the necessary protection against ankylosis. We were unable to rotate the patella, as is usually done in these cases.

We have little fear of necrosis of the external skin-flap. Formerly I used to make a U-shaped flap. We had quite a good deal of trouble then, because occasionally the tip of the flap became necrotic. Since we have been making these flaps we have had no trouble at all. There was very little bleeding in this case and we are ready to close the skin incisions with deep catgut and superficial horsehair sutures. The iodin used to sterilize the skin will be washed off with alcohol and the wounds dusted with bismuth subiodid powder. The former prevents an iodin dermatitis from occurring; the latter is merely a desiccating powder. Gauze dressings wet with alcohol containing 1 per cent. of phenol will be applied, and over this a plain sterile gauze dressing, held in place with roller bandages. The leg will be flexed slightly,—kept in the Phelps position,—and a 20-pound Buck's extension applied.

[Date of operation, April 24, 1913. The first dressing was made on the tenth day after the operation. The wound had healed by primary union, but a hematoma had formed on both sides of the patella. Eight drams of sterile, non-odorous, clear bloody fluid were removed from each side by aspiration, and the point of entrance of the needle sealed with collodion. The joint was strapped with two-inch strips of plaster, 18 inches in length, extending from above downward, crossing the patella in figure-of-8 fashion, but not going around the joint. The strips were placed alternately, one from above and external downward to below and internal, and the other from above, internal downward to below and external, making a plaster spica on the anterior surface of the joint. At the second dressing, made on the fourteenth day, no fluid was present in the joint nor in the tissues around the joint. The patient has felt entirely well since the operation, with no pain in the joint. Motion is returning slowly. Ten weeks after the operation he was able to flex the knee voluntarily to an angle of 30 degrees. In the Knokke and Dresler machine he is able to flex the knee to right angles. He has no pain in the joint, and bears his full weight on the leg without discomfort.—Ed.]

POSTSACRAL DERMOID

HISTORY

Patient, male, single, aged twenty, enters hospital on account of slight pain and constant irritation around anus from a discharging fistula. Two years ago he says he had a small, elevated, very sore mass in the skin to the left of the anus. It was very painful for two weeks, when it ruptured and discharged a foul-smelling pus. A sinus formed, and since that time it has continued to discharge. He was operated on unsuccessfully for fistula in ano in October, 1911. The sinus is still discharging. Two months ago (February, 1913) he had another abscess over the buttocks and two more to the left of the anus. These abscesses ruptured, discharging a bloody pus. There is still a considerable discharge, and the skin around the anus is excoriated. Past and venereal history negative.

COMMENTS AND OPERATION

Dr. Murphy (Operation, April 15, 1913): This case, judging from the history and the appearance of the lesion, looks like a postsacral dermoid, that is, a fetal remnant behind the sacrum. That fetal remnant became infected, an abscess formed, it ruptured, and ever since there has been present a discharging sinus. If it is a postsacral dermoid, there is only one way to cure it, and that is to dissect out the sac. No other treatment does any good, because the sac is lined with epithelial cells, and these continue to secrete fluid, which becomes infected. Draining would not benefit him at all. The lining of the sac, the part that is doing the mischief, must be dissected out.

There is one feature in this case which makes me think that this might be an anal fistula, and that it is in the line of the incision from the former operation, which looks as though it had been a fistula. Did that old sinus extend into the rectum? I do not see a scar near the anus. The scar seems to be about half an inch from the anus. That looks like the typical invasion that occurs with postsacral dermoids. All postsacral dermoids are sequences of congenital defects in the neuro-enteric canal. They are tubulodermoids, arising from a tube lined by mucous membrane—the postanal gut or from involuted skin in this case—which takes the place of skin in the wall of the cyst, and the contents consist of the pent-up secretions of mucous glands. There are two conditions which we must consider here. One is the postsacral dermoid, the other is tuberculosis. However, I am inclined to the belief that it is a dermoid. You will note that the probe passes from the opening half an inch from the anus upward for four inches toward the posterior surface of the rectum. An anal fistula would not burrow in this direction nor would it appear in this position. We will expose the sac and dissect out its lining. These incisions in this region are very slow in their process of repair.

We will make a longitudinal incision directly over the location of this dermoid, passing to one side of the fistula, going through the skin, superficial and deep fasciæ, into the sac. There is some hair. That is the rule for these dermoids. They always contain some fetal remnant—hair, teeth, fragments of bone. The sac is lined by a granulation tissue. There is only hair in this dermoid. I must make sure now that there is no other track leading off from this sac, because if we do not excise all the dermoid, it will fill again. We will dissect out the entire sac, so that there will not be a recurrence of this trouble. What an extensive procedure that is when you start after it! We did not penetrate into the bowel at any time.

The sac being now removed, we will close the wound first with silkworm-gut sutures. These sutures will immobilize the tissues. A rubber tissue drain will be placed in here, extending from the upper angle of the wound downward and out through the lower angle. The usual alcohol dressing (plus 1 per cent. phenol) is applied.

The important factor in this case is the differential diagnosis.

Pilonidal cysts and postsacral involutions of a remnant of the neuro-enteric canal are commonly treated by opening and drainage, by curetment, by cauterization, by iodin injections, etc. These methods are all ineffectual. These sacs are lined with epithelial cells, and only a complete dissection of the sac lining produces a cure. If any portion of the lining remains, or if diverticula are overlooked, recurrence of the suppuration is inevitable. Post-procteal cysts or dermoids are also of embryonic origin, but occur in front of the sacrum and behind the rectum. We had such a case not long ago. They require the same treatment—complete dissection of the sac and its removal.

[Healing progressed better than expected. The patient left the hospital after four weeks with the wound completely healed. —Ed.]

PSEUDARTHROSIS OF SHAFT OF HUMERUS —ANKYLOSIS OF ELBOW—WRIST-DROP

HISTORY

Patient, male, aged thirty, comes to hospital on account of inability to extend the left wrist and to flex or extend elbow, and a false point of motion about two inches above the left elbow. The trouble dates back to September 2, 1912, when a gun slipped to the ground and was discharged, the bullet entering the left arm on the anterior surface, just above the elbow, and coming out on the external surface. The wound bled freely. The brachial artery could be seen pulsating. He had severe pain immediately after, and a false point of motion two inches above the elbow. On account of the extreme hemorrhage he was given a subcutaneous saline infusion. He was operated on at 11 P. M.—fifteen hours after the injury. He was told that the humerus was shattered; that three-fourths of an inch of the shaft was gone, and that the external condyle was split off. A Lane plate was put on the fractured shaft; drainage-tubes were inserted, and the arm was placed in a splint at right angles. There was not much discharge from the wound the first three days, but after that there was a great deal of discharge for nine weeks. During the first six weeks he suffered a great deal of pain in the arm. He does not remember having any distinct pain in the elbow. His temperature was elevated during this time. When the Lane plate was removed at the end of the ninth week he had a false point of motion two inches above the elbow. The arm was put in a cast during November and December, 1912. When it was taken off in the latter part of December he had no union at the seat of fracture and the elbow was stiff. Neither could he extend the wrist. At present there is a small sinus on the external surface of the arm, about two inches above the elbow. One near the joint closed recently. He has wrist-drop and an ankylosis of the elbow.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 16, 1913): He has a wrist-drop, but has full function of all the muscles of the forearm supplied by the median and ulnar nerves. He has a paralysis of all the muscles supplied by his musculospiral. I do not think it is possible to do anything with the musculospiral, because of the extent of destruction of the nerve and soft tissues on the external surface of the arm. A great deal of tissue was destroyed, and in all probability there is so much of the musculospiral nerve gone that we cannot approximate the divided ends. We can, however, remedy the wrist-drop.

The ulnar nerve supplies the lumbricales and interossei muscles. They act, first, as extensors, and, second, as flexors, having a double action. When his hand is down, he can extend his fingers and also flex them slightly. His flexor carpi ulnaris and flexor carpi radialis are intact. We will do what we have done before. We will implant the flexor carpi ulnaris into the extensor carpi radialis to produce extension of the wrist. He will learn to use that flexor muscle as an extensor.

The principal question in the management of this case is what we are going to do with the elbow. He has an ankylosis of the elbow and a pseudarthrosis about two inches above the elbow at the seat of the fracture. It would be very easy for me to do a bone-transplantation and get a union of the humerus in this case, but he would still have a bony ankylosis of his elbow. He would be worse off than before, because he would have a stiff arm. The best treatment for this man will be to make a controllable pseudarthrosis above the elbow, so that he can use his arm. The forearm is in hyperextension and cannot be flexed. He is a doctor, and does a great deal of obstetric work, so that he must have the flexion of his forearm. Therefore, if we can give him a false joint above the elbow and restore the power of extension in his wrist, he can get along very well. If he did not have the ankylosis of the elbow, we would do a bone-transplantation in the humerus, but under existing circumstances the best we can do is to make a controllable pseudarthrosis at the site of the non-union of the humerus fragments.

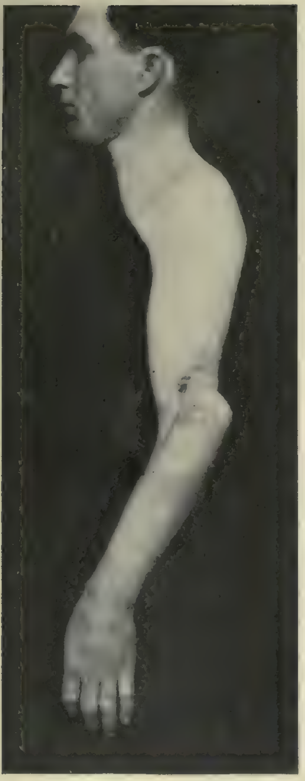

Fig. 155.—Fracture of the humerus. Pseudarthrosis. Position of arm before operation, showing the point of non-union just above the elbow.

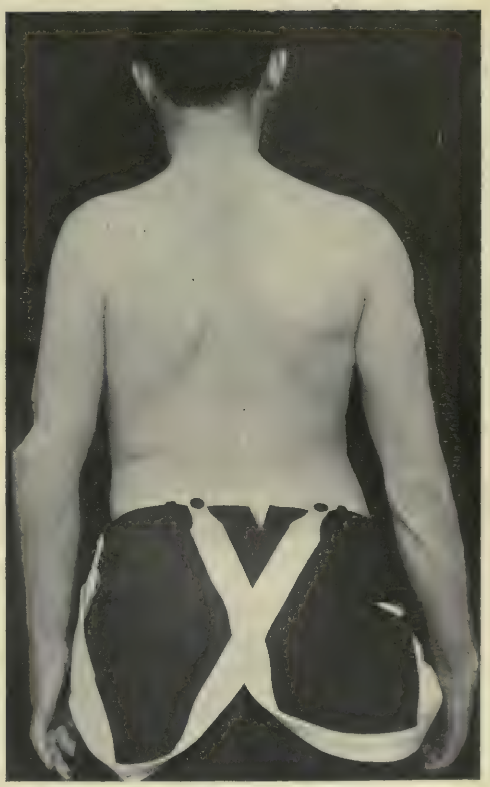

Fig. 156.—Fracture of the humerus. Pseudarthrosis. View of arm from behind before operation.

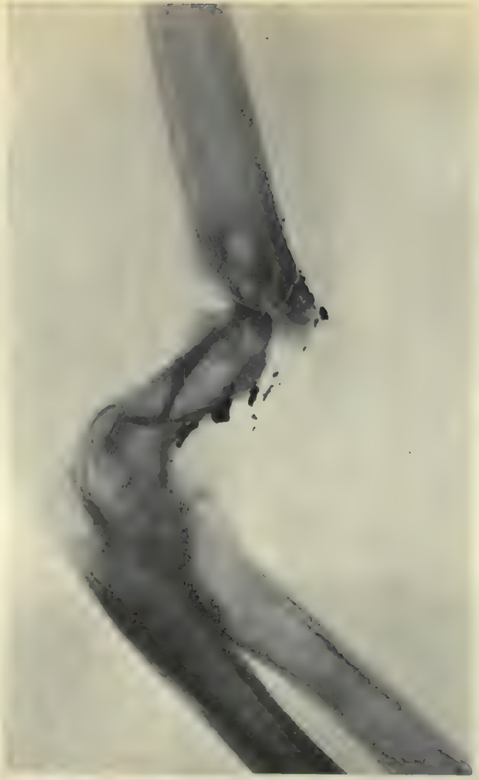

Fig. 157.—Fracture of the humerus. Pseudarthrosis. Lateral view made before operation. Bony ankylosis of elbow involving all bones, with joint flexed at right angle, non-union of fracture, with a fibrous ankylosis—a typical pseudarthrosis. The black shadows are remnants of the shot which caused the injury.

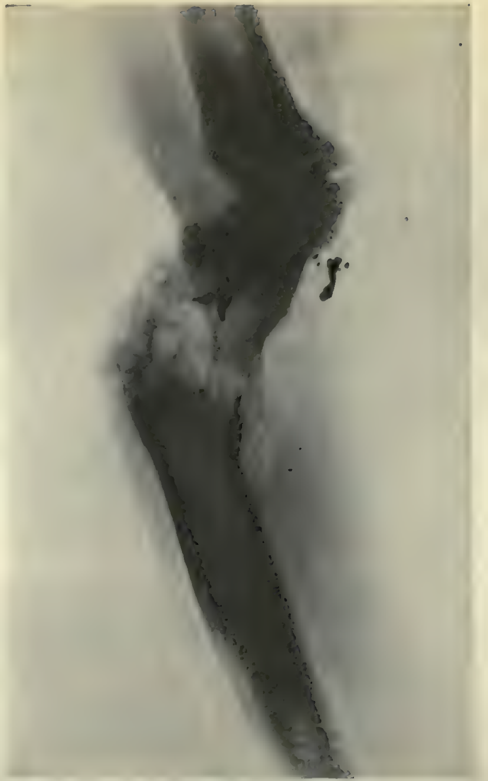

Fig. 158.—Fracture of the humerus. Pseudarthrosis. Another lateral view before operation taken from opposite side.

A suppurating sinus is still present, which we dislike very much in doing this type of work. Remember that the artery was exposed at the time of injury. It was pulsating, but was not ligated, so that we expect to come right down on that in this operation, and we may possibly have to ligate it.

We will make a longitudinal incision on the external lateral surface of the arm, extending upward from just above the external condyle about four inches. We will have to be very careful not to injure the artery. There is the upper end of the musculospiral. It is frayed out and fibrous. Testing the nerve proximally with the excitor, we find that it is functionating. The lower end is not in view. We will be very careful not to injure the proximal portion of the nerve. We will next free the upper end of the lower fragment, preserving its periosteum for future use as an interposing tissue. The lower fragment is behind and internal to the upper fragment, as was shown by the skiagram. I want to see if there are any fibers of the distal end of the musculospiral nerve remaining intact, and then throw them into this field, if possible. There are none left, however. The faradic current shows no response. If there was one fiber left of the musculospiral, we could make a union of the divided ends of the nerve, and in all probability function would be restored to the muscles supplied by it below the elbow.

We have no anatomic landmarks here to go by at all. We are just feeling our way along. I wish to preserve the periosteum, as otherwise there will be a reproduction of bone here subsequently that will be detrimental. There is the line of fibrous union of the two bones. I will separate this fibrous union, preserving the periosteum. Now we are beginning to see a fairly clear outline of the ends of the two bone fragments. There is some muscle tissue between these fragments. That is the reason he had a non-union.

By very careful dissection we have succeeded in freeing the ends of both fragments. The median nerve is now in view. I have to take off a portion of the lower fragment so as to make a socket in which the end of the upper fragment can slide. It will be a sliding joint. I will square off the end of the lower fragment first, and then take out a quarter round piece from above downward and forward on its anterior surface. I am going to utilize the periosteum as an interposing flap, because I have no other tissue that I can use as a flap on account of the cicatricial tissue which has formed as the result of the injury and the previous operation. The periosteum will cap the upper end of the lower fragment. The lower end of the upper fragment will be rounded off to fit the groove in the lower fragment. Now you see what I have done. What I expect to get will be a controllable sliding joint. Judging from the result secured thus far, he will have a good joint here.

The fibrous tissue around the ends of these fragments is as firm as an ordinary capsule, and it will tend to keep them immobilized; that is, keep them from sliding to the right or left during the process of repair. I have the artery between my fingers. It is pulsating nicely, so that the circulation is not going to be impaired at all. There is the musculospiral nerve. We are leaving that open—open to conviction, that is; later we may endeavor to do something with it, but I doubt it.

There was practically no bleeding at all. We did not have to ligate a single vessel. The wound will be closed with catgut, silkworm-gut tension sutures, and horsehair to approximate the skin-edges. The usual alcohol and 1 per cent. phenol gauze dressings will be applied. The arm will be kept at a little less than right-angle flexion by means of a plaster-of-Paris cast extending from the shoulder to the finger-tips.

If we get a pseudarthrosis there, as we hope we will, he will be able to put that hand through all the range of motion that is essential for its usefulness.

[Nothing untoward happened after the operation. The patient suffered very little pain and discomfort. The stitches were removed on the sixteenth day, when the first dressing was made. Primary union. Passive motion was instituted at this time, and May 20th, less than four weeks after the operation, the patient could bring his finger-tips to his nose. The pseudarthrosis was all that had been expected. Eleven weeks after the operation the patient had a good range of motion in the new joint and had more use of his arm than he had had since the injury.]

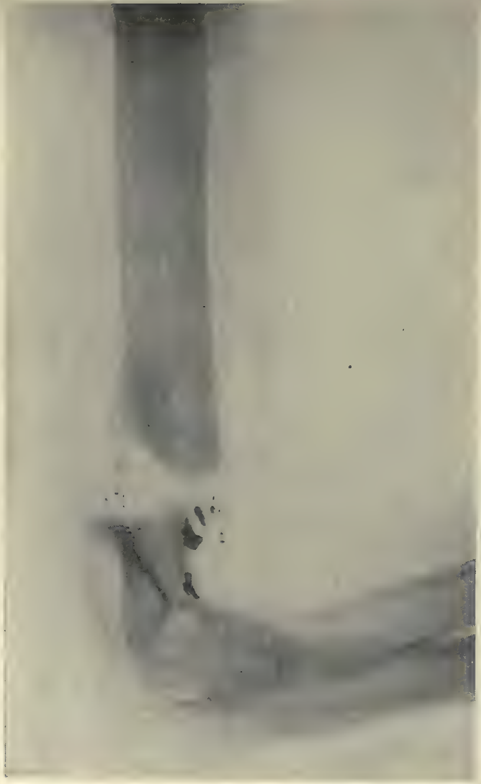

Fig. 159.—Fracture of the humerus. Pseudarthrosis. Lateral view made four weeks after the operation. The patient had a freely movable flail-joint at the site of the fracture, the upper fragment sliding in a groove made in the lower fragment.

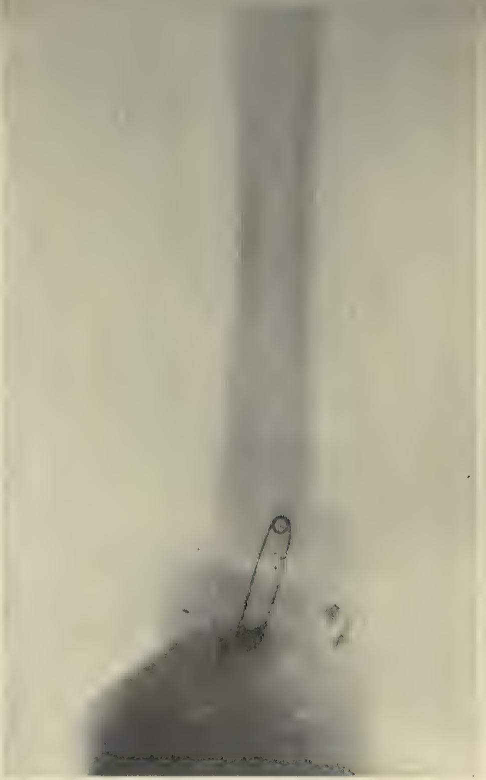

Fig. 160.—Fracture of the humerus. Pseudarthrosis. Arm viewed from behind, showing the form and position of the flail-joint. The safety-pin is in the dressings.

BONY ANKYLOSIS OF JAW, WITH INTERPOSITION OF FLAPS FROM TEMPORAL FASCIA

HISTORY

Patient, male, aged twenty-four, comes to hospital on account of limited motion in jaw. The condition dates back eighteen years (1895), when he was kicked by a horse on the left side of his jaw, just posterior to the mental angle. He sustained a compound fracture at this point. The wound suppurated and discharged pus for about two months, when a sequestrum of bone or a tooth (he does not know which) came out through the opening. The wound then healed. Immediately after the accident the patient also had a discharge from the right ear which persisted for some time. He never had any pain. Following this he had a complete fixation of his lower jaw. Two upper teeth, the right canine and the bicuspid, were removed. He eats only soft foods. Past venereal and family histories negative.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 17, 1913): What is the matter with him?

Intern: He has an ankylosis of the jaw.

Dr. Murphy: What makes you think so? If it is an ankylosis of the jaw, what kind of an ankylosis is it?

Intern: He cannot open or close his jaw, or move it laterally. It seems to be a bony ankylosis.

Dr. Murphy: Ankyloses of the jaw may be divided into—First: Intra-articular conditions; it may be a fibrous ankylosis. Second: It may be an osseous union between the bones—a bony ankylosis. Third: It may be due to periarticular conditions. Fourth: It may be due to extra-articular muscular or cicatricial fixation on the side of the face or cheek or a binding together of the alveolar processes. These are the propositions which one must consider in cases of ankyloses of the jaw. Remember that this man was kicked by a horse on the left side of his face, and he also had a discharge from the right ear. He has no scar on that side of the face. He told me that he had a suppuration in the right ear and a suppuration at the angle of the jaw. Either of these conditions might tend to produce an ankylosis of the jaw, but they would be of two distinct varieties. He had a metastatic infection in the right mandibular articulation and the abscess opened into the ear. He had no extension of the infection throughout the entire length of the mandible from the seat of the fracture, as one sees in mouth infections, typhoid infections, or phosphorus necrosis, which may involve the whole jaw from the symphysis up into the mandibular articulation, so that the head of the bone becomes necrotic and is exfoliated. In those cases you can take out the whole jaw as a sequestrum. There is no evidence that he has had such an infection. We are convinced that he had an injury on the left side of his jaw; then he had an infection on the right side, in the mandibular articulation, extending into the temporal bone. This is a double joint. Evidently both cavities were involved. That is what produced the ankylosis.

Infections of the middle ear penetrate in four different directions, one of them being the most common cause of infections in the mandibular articulation. These routes are: First, the infections that pass backward into the mastoid; second, those that pass through the posterior part of the petrous bone into the posterior cerebral fossa; third, those that penetrate the attic of the ear and form within the cerebral fossa and rupture externally, just above the ear, or that burrow forward and extend into the mandibular articulation; fourth, where the abscess burrows forward under the base of the zygomatic process and infects the mandibular articulation. What happened here? Did he have an infection of the cheek unassociated with the trauma? There was no such infection into the mandibular articulation, as far as we are able to ascertain. This is not the usual course—it is an exceptional one. The other conditions that produce ankylosis

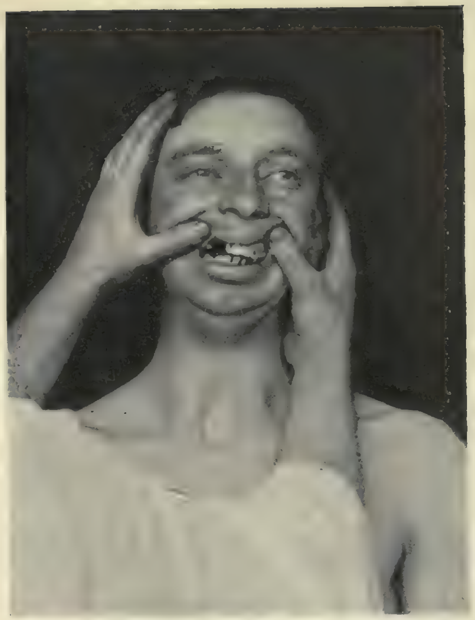

Fig. 161.—Ankylosis of jaw—before operation. The jaws are tightly closed. The patient fed himself through the gap in his denture on the left. The jaw is drawn toward the affected side—the right.

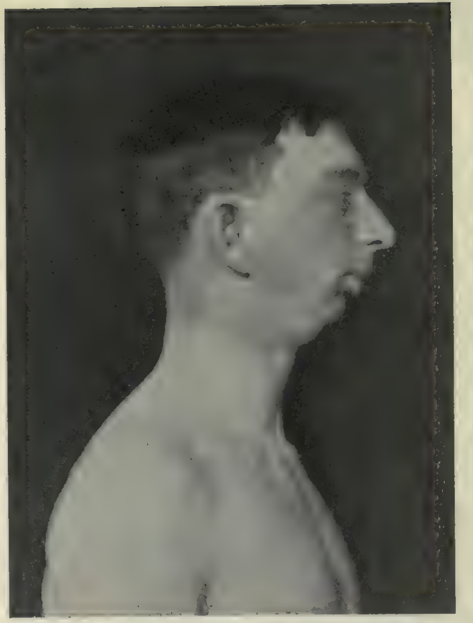

Fig. 162.—Ankylosis of jaw. Profile view before operation, showing the retraction or shortening of the lower jaw typical of all these cases of long standing.

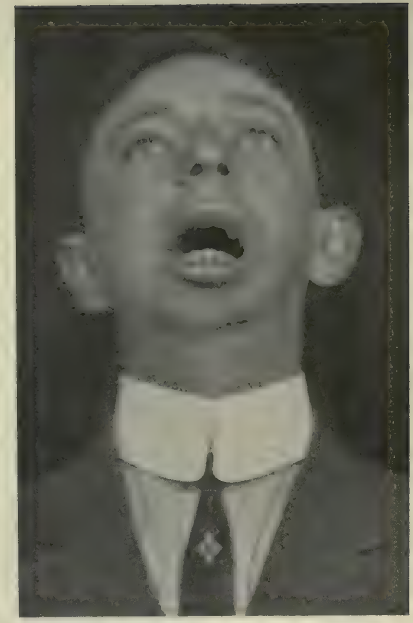

Fig. 163.—Ankylosis of jaw. Photograph made about four weeks after the operation. The patient could separate his jaws voluntarily about an inch. Perfect freedom of motion.

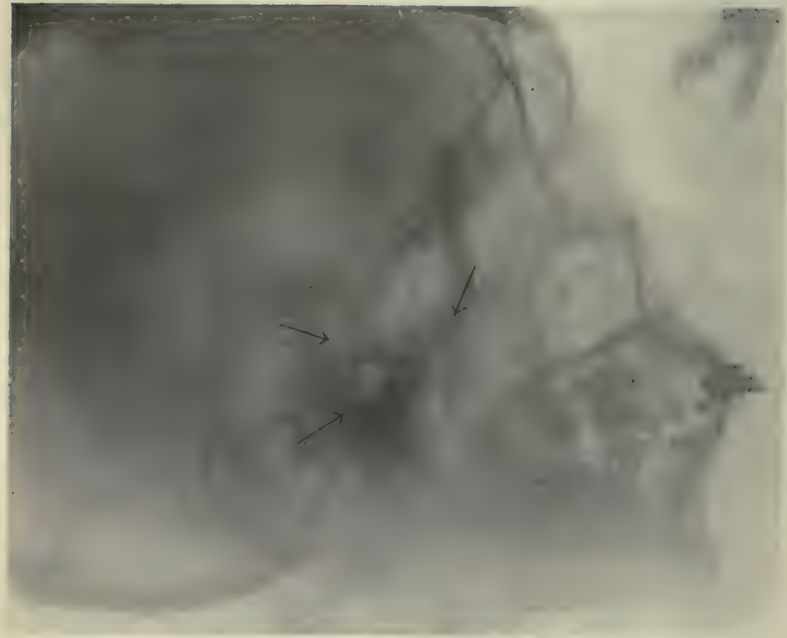

Fig. 164.—Ankylosis of jaw. Skiagram made before operation, showing complete obliteration of the right temporomandibular joint. The bony ankylosis extended clear across to the coronoid process.

are the infections that occur about the mouth, the sloughing of the mouth in typhoid and scarlet fever, noma, etc.

We had a case in which there was no trouble at all with the mandibular articulation. The patient had had a sloughing of the cheek following a typhoid infection which produced an enormous destruction of tissue, with the formation of a cicatrix. The jaw was absolutely fixed.

We had another case of an infection of the right temporomandibular joint, with a deformity on the left side of the face. The boy's parents told us that the suppuration had been on the left side. When we went in to operate we found a bony ankylosis on the right side instead of on the left. We did an arthroplasty in that case, interposing a flap taken from the temporal fascia. He had not opened his mouth since he was seven months old. When he left the hospital he could open it one inch. (See December, 1912, CLINICS.)

In making a diagnosis as to the side affected, we have learned, from our clinical observation, that it is *not* the side on which there is a deformity, but the opposite side, the side on which the jaw is fixed either by a fibrous or bony ankylosis.

Ankyloses of the jaw are, on the whole, very difficult cases to handle. In the last case, one of fibrous fixation, extra-articular, we resorted to a new plan for its management, the interposition of mucous membrane flaps taken from the palate and floor of the mouth. It worked out very nicely. (See April, 1913, CLINICS.)

This patient today has been operated on before. That makes a second operation difficult. I do not know whether the surgeon ligated the temporal artery. We will expose the articulation through a two-inch incision, beginning at the lower level of the tubercle of the zygoma, half an inch in front of and on a line with the external auditory canal and extending upward into the hair. By retracting the wound margin and elevating the tissue with a periosteotome we are at the articulation—not that we recognize it as an articulation, but we recognize a mass of fibrous tissue and an elevation that feels like the head of the mandible. Here is fibrous tissue resembling the capsule of the articulation—hard and firm. There is no motion at all in the articulation. Now we have exposed here the head of the bone. I will take off the periosteum of the head and see if we can determine whether there is a bony or just a fibrous ankylosis on this side. It is a bony ankylosis. The important point in this work is to get a good exposure. The ankylosis runs clear forward to the coronoid process, uniting the mandible to the zygoma. The line of demarcation between the mandible and the zygoma is distinguishable.

I have it fairly well freed. Now we will divide the ankylosis with a narrow, straight chisel and remove the head of the mandible. I will have a space of at least one-third of an inch which we will fill in with the interposing pedicled flap of temporal fascia. The real point of fixation was at the coronoid process. The coronoid process was united to the under surface of the zygoma so that it fixed the mandible firmly to the zygoma. Next, we will raise a flap of the aponeurosis and fat of the temporal fascia, about two inches in length and an inch in width, base directed downward, drop it over the zygoma and into the glenoid fossa, so that it is interposed between the two bones entering into the formation of the temporomandibular articulation. That will prevent the recurrence of the ankylosis. The tip of the flap will be fastened in place with a few tacking catgut sutures and the base fixed to prevent retraction—also with two sutures. That completes the operation, except for the closure of the skin incision. This is done with horsehair. The wound is dusted with subiodid of bismuth and sealed with collodion gauze. There was very little hemorrhage. No other dressing will be applied. The jaw moves quite freely, but there is some fibrous fixation of the opposite side. This will be overcome by stretching later.

[The day after the operation the patient had free motion in the jaw. This increased rapidly. When the skin sutures were removed on the tenth day, he was able to open his mouth one inch. A week later the fibrous fixation of the left temporomandibular joint was overcome by prying open the mouth under anesthesia. The patient left the hospital on the twenty-second day. He could open his mouth an inch and a half. The result was ideal. There was no scar on the face that was perceptible, and no depression where the temporal flap had been deflected downward.—Ed.]

UNUNITED FRACTURE OF THE TIBIA: REMOVAL OF SILVER WIRE; BONE TRANSPLANTATION; NON-UNION OF FRACTURES—CAUSES

HISTORY

Patient, male, aged thirty-six, comes to hospital on account of pain in the calf of the right leg and deformity (bowing outward on walking) about five inches above the ankle. Trouble dates back to October 12, 1911. At that time the patient was kicked by a horse on the right leg, five inches above the ankle. He did not attempt to stand on the leg, because the foot had turned out and its inner surface was flat on the ground when he was sitting with the leg extended. The tibia and fibula were fractured. He had some pain when the leg was lifted up. Pain was severe for the next two weeks. The leg was dressed in a box splint with an extension applied for two weeks. The skiagram showed that the bones were in good apposition. A plaster-of-Paris cast, including the foot and the thigh, was applied, and left on for two months. When the cast was removed, there was no deformity, but there was a false point of motion five inches above the ankle. Another cast extending above the knee and including the foot was put on, and the patient was permitted to be around on crutches. This cast was removed after six weeks. There was still some motion at the seat of fracture. From that time he walked with a cane. The leg was slightly bowed anterior at the site of fracture, and he had a great deal of pain on walking.

A skiagram, taken in July, 1912, showed a non-union of the tibia. The fibula had united. August 5, 1912, an operation was performed at his home city. The ends of the tibia were exposed, freshened, and wired with silver wire. A plaster cast was put on and left on until the latter part of September (six to seven weeks). Union was not very firm. He had a leather support made for the leg and wore a high shoe. About the middle of November he began walking without any support. He still had pain at the site of the fracture. Wound healed by primary union. One week ago (April 5, 1913), patient noticed a small raised red spot above the point of wiring. This has increased in size slightly since then. There is still a false point of motion at the site of the old fracture, with pain at this point on walking. Previous history negative.

COMMENTS AND OPERATION

Dr. Murphy (Second operation, April 26, 1913): The first operation (April 12th) which we performed on this man merely consisted in exposing the fracture, taking out the silver wire, and removing all the granulation tissue. Because of the suppuration that had taken place around the wire we would not make a transplantation at that time. One must have an absolutely aseptic field before attempting to do a bone transplantation. That is the first essential to success in this work.

Now we come to the proposition that nineteen months after the injury this man still has a non-union. The skiagram shows that there is a splendid apposition of the fragments. We were informed that the apposition was good before the fracture was wired. The wire, therefore, did nothing to approximate the bones, and the irritation of the ends of the fragments did nothing toward the production of bony callus for the union of the fragments.

The question that arises now is, Why was there this failure of union? Why are these cases occurring more frequently now than formerly, either with or without operative procedure? I have discussed this in one of the recent clinics, but I wish to go over it again as it is my impression that we fail to do the best we can in our treatment at the time the fracture occurs. We do not fail in the fact that we do not get good apposition of the fragments, because the bones are usually put in apposition, but we fail in the fact that while we get a fairly good apposition, we produce a too perfect immobilization for the best osteogenesis. Absolute immobilization is not conducive to the greatest repro-

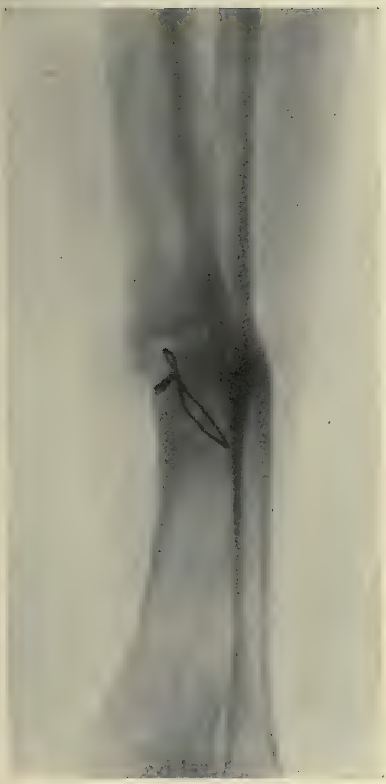

Fig. 165.—Ununited fracture of right tibia. Lateral view, showing a fairly good apposition of the fragments with apparent bony union. There is only a slight excessive callus visible. The silver-wire fixation suture passes through both tibia fragments.

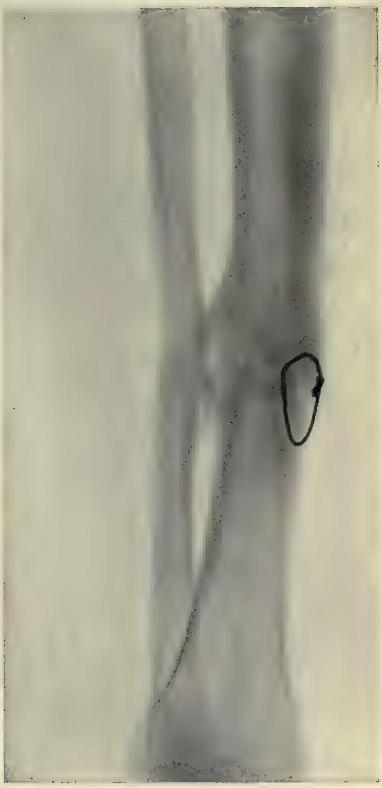

Fig. 166.—Ununited fracture of the tibia (anteroposterior view). Alinement is perfect; apparent bony union. The excessive callus is now seen to be considerable in amount, especially on the outer side of the tibia. The silver wire is on the internal aspect of the tibia, passing through both fragments.

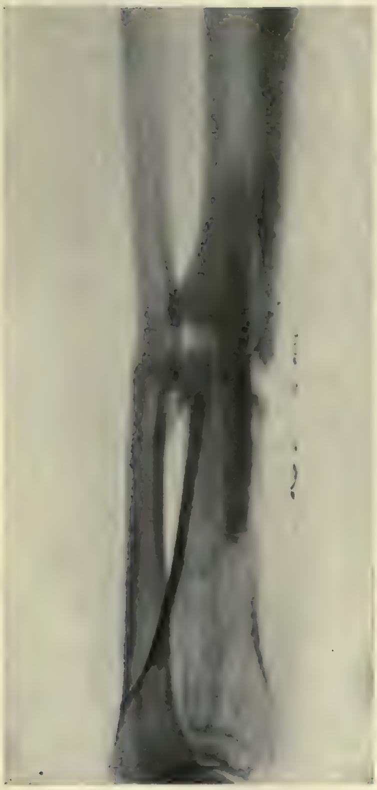

Fig. 167.—Ununited fracture of the tibia. Anteroposterior view, made about four weeks after the transplantation. The transplant in the tibia is clearly visible. The leg was quite firm, showing that osteogenesis and bony union were progressing rapidly. The leg is perfectly plumb.

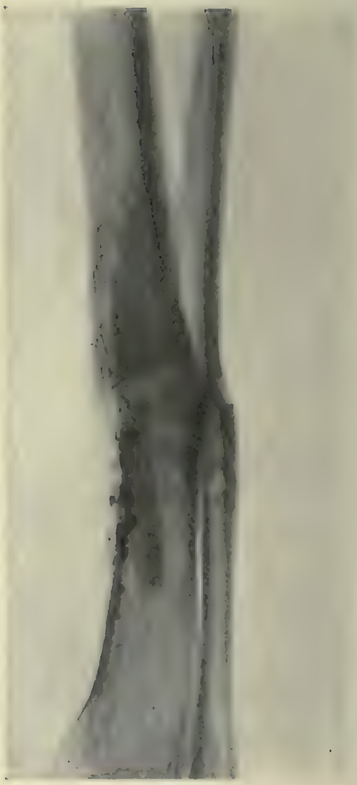

Fig. 168.—Ununited fracture of tibia. Lateral view, made about four weeks after the transplantation. The transplant shows less distinctly than in the previous picture. Bony union appears to have occurred.

duction of bone or callus. Twenty-five or thirty years ago we practically never had a case of non-union of a fracture. In the period preceding the use of plaster casts in fractures we rarely ever had a non-union. When I was in the Cook County Hospital late in the seventies and early eighties we had the largest fracture service in the city. I recall only one case of non-union. In the ten years' service in the Alexian Brothers Hospital we rarely ever had a non-union. We had cases of retarded union, but rarely a non-union. Today cases of non-union are so common that I do not know what we are going to do.

What we should do, it seems to me, is this: If you are going to immobilize with casts and fixation apparatus, then the patient should be put in ambulation—start him walking, keep up a constant irritation of the ends of the fragments. That will stimulate union. It favors osteogenesis. You will notice in the skiagram of this patient that there is just the slightest effort at the production of a periosteal callus between the tibia fragments, but there is no effort on the part of the medullary callus to span the line of fracture. Mr. Lane is quite correct in his statement that the metal bone-plate should not be used for non-united fractures. You must do something else to conduct the osteogenetic process across the line of fracture. That is what led us originally to our present plan of treatment—the transplantation of bone. The essential point in that operation is the necessity of securing and maintaining the strictest asepsis. If you will span the gap between the fragments by means of an auto-bone transplant, you will secure a union.

The fibula in this case has united. Therefore we will implant a fragment of bone into the tibia only. We will take it from the opposite tibia. Contrary to the treatment employed in the cases of recent fracture, you must have perfect immobilization for the first six or eight weeks, as here you wish to have the Haversian vessels pass from the living bone-ends into the transplant. If friction or motion is kept up, that does not occur, and connective tissue is formed around the transplant and union is retarded if not prevented entirely. The leg is put in a plaster cast and the patient instructed to walk on crutches.

We will make the incision through the old scar over the tibia, and by retracting the soft parts, the old fracture is well exposed. There is the line of separation of the bone. I will elevate the periosteum from the ends of both fragments over the position where I am to make the implantation, because it will be of service in inducing the production of new bone. The ends of the fragments will be freshened and then we will remove a rectangular piece of bone several inches in length from each fragment with the Zapffe motor buzz-saw. That will facilitate placing the transplant. The medullary canal of each end will be reamed out with our special reamer to receive the transplant. The upper fragment will be reamed out for a distance of 2½ inches, the lower, one inch. We will insert the transplant into the upper fragment first, and then drive it down into the lower. We are ready to remove the transplant. This leg will be covered with sterile towels in the mean time.

We will take out our transplant from the crest of the opposite tibia, the skin of which was prepared before we began the operation. The incision is made half an inch to the outer side of the crest of the tibia, so as not to have the line of skin union over the crest subsequently. The muscle fascia is divided and the muscle retracted so as to expose the bone. I am going to split the periosteum, leaving a strip on the piece of bone to be removed. I do not believe that this is essential to success, but we have not had any failures with our present technic, which preserves the periosteum, so why should we change our methods? We are not going to lessen the chances of success by any needless experimenting. We believe, with Macewen, that the periosteum *per se* does not possess any osteogenetic powers. It is solely a limiting membrane. In at least one of our cases it was a detriment, as shown by the skiagram.

Visiting Doctor: Do you put a constrictor on the leg?

Dr. Murphy: No. We do not consider it necessary—in fact, we believe it is detrimental. There is little bleeding. Remember that there is danger in handling every instrument; that every trauma you inflict is injurious to the nervous system. We nick the bone transversely to mark the length of the transplant, and we remove it from the crest of the tibia with the Zapffe motor saw. That is done in a few minutes—much more rapidly than with the chisel and with far less labor and effort. In this case we remove a fragment 4 inches long by 5/8 by 3/8 by 1/2. We always grasp the transplant with two sequestrum forceps. With two forceps on it there is so much less danger of the transplant slipping and flying out of your grasp. We leave the periosteum on the transplant, not because we regard it as a necessity, but because it may be of advantage.

The wound in this leg will be closed by reuniting the muscle fascia with catgut and the skin with horsehair. It will be dressed with gauze saturated with alcohol and 1 per cent. phenol, held in place with a roller bandage. The wound is first dusted with bismuth subiodid and sealed with collodion gauze.

Now we will place the transplant, inserting it first into the upper fragment and then into the lower. The grooves I made are a great help. Now we have succeeded, you see, in putting that implant in across the line of non-union. In driving this transplant into the upper fragment, I have frequently seen the periosteum rubbed off, but that did not, apparently, interfere in any way with bone reproduction. Now we are ready for the closure, which will be made in the same manner as in the opposite leg.

The rôle which that transplant plays in the regeneration of bone is one of conductivity of osteogenetic elements, not reproduction. There is some difference of opinion as to whether that is true or not. That does not matter a whit to the surgeon, however. The fact is that it favors or forces union. Whether it produces union because of the osteogenesis that is inherent in it or by the osteoblasts which travel through it from the bone above and below by the chemotactic or other force, does not matter either to the patient or to the surgeon. Just what rôle it plays we are trying to find out for the purpose of furthering work in transplantation. We have laid down a law, however, and that law must stand until we find something to disprove it, namely, that the transplant must contact with living bone, at least at one end. It is being said now that you can reproduce bone from bone without contacting it with living bone; that a fragment of living bone placed in muscles will reproduce *permanent* bone. We do not believe that it will. A number of articles on that subject have been published, particularly in the German literature, where bone was produced in that manner and continued in evidence at the end of six or eight months. That observation has no value because it takes bone a longer time than that to absorb. If it is still growing at the end of sixteen or eighteen months, then you can say it has osteogenetic power. Many years ago I took the second phalanx of the thumb and transplanted it into a nose in a case of saddle-nose. I showed it for months afterward as a beautiful result, but in fourteen months it had all disappeared, meaning that there was absorption but no osteogenesis. We had left the periosteum and cartilage on the bone, and there was, therefore, no means of having a freshened bony surface of the transplant contacting with living bone. It was absorbed, leaving only a mass of connective tissue remaining. Slowly but surely it all disappeared. That is what we expect.

Concerning the tibia on the other side from which we removed the transplant, that gap will fill in rapidly. We have had to take out a second piece of bone from the same area, and we had no trouble in securing it, showing that the tibia was reproduced.

The great precaution to observe about this work is that it should be strictly aseptic. If that wound were to suppurate, union may not occur, but still he could have a union although the transplant fragment would have to be removed. But union could take place on the side by the mechanic irritation of the transplant; in fact, it would be stimulated by it.

VISITING DOCTOR: Would you get the same result if you transplanted bone from a preserved specimen?

DR. MURPHY: About the transplanting of tissue from another individual: Only a small percentage of the homo-grafts survive, while in our work 100 per cent. of the auto-grafts have survived. In Tuffier's work on the ovary, which I like to cite because it exactly corresponds with our experience with bone, he showed that when he took an ovary from the same individual and transplanted it from the peritoneum to the connective tissue just external to the aponeurosis of the rectus muscle, a large percentage of these ovaries lived. When he took an ovary from one woman and put it on the rectus muscle of another, then only a small percentage lived. When he took it from one white woman and put in a black woman, or from a black to a white woman, they all perished, showing that the mere color or family plays an important rôle in the transplanting. We have found that if you take a piece of cow's bone or ox bone and transplant it into the human that it merely acts as a mechanic stimulant. It plays no osteogenetic rôle. If you transplant a piece of bone from one person to another, it lives in a considerable number of cases. If you transplant bone from one portion of the patient's body to another and contact it with living bone, at least at one end, 100 per cent. are osteogenetic; that is, they are osteo-conductive. They "live" and fulfil the purpose for which they were intended.

The ordinary plaster-of-Paris cast will be put on in this case, extending from the toes to the groin. It is split with the Gigli saw (placed on the leg before the plaster is applied) as soon as the cast has hardened.

[The subsequent progress of the case was uneventful. Stitches removed on fourteenth day. Primary union. Leather cast substituted for plaster cast after fifth week. Patient on crutches. The skiagram made twelve weeks after the operation shows a good apposition of the fragments, with a perfectly firm bony union.—ED.]

OLD UNUNITED FRACTURE OF THE TIBIA—TRANSPLANTATION OF BONE

HISTORY

Patient, male, aged thirty-six, comes to hospital on account of inability to bear body weight on left foot and a false point of motion about three inches above the ankle. August 6, 1911, he fell a distance of 30 feet into a pit, and landed on his left foot. He fell over, but did not become unconscious. On looking at his leg, he saw that the tibia was protruding through the skin, midway between the ankle and the knee. He had no chills, but a temperature of 102° F. on arriving at hospital shortly after the accident. August 9th, three days later, he was operated on and the fragments were approximated with silver wire. A drain was put in and a cast applied. The wound suppurated and discharged sequestra of bone. At the end of three months the silver wire was removed. The wound closed at the end of five months. Since that time he has had to wear a steel support because the leg will not sustain his weight. He has considerable pain when walking. He had typhoid nine years ago, and a Neisserian infection in 1898.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 19, 1913): The skiagram shows an ununited fracture of the tibia, with a large exostosis on the outer side of the lower fragment, extending upward several inches above the lower end of the upper fragment. He has a union of the fibula which we are not going to refracture because the leg is fairly straight. There is the same condition here, although somewhat less in degree, that there is in the arm, namely, the tendency to adduction of the lower fragment, that is, the foot is turning inward. This is a case of non-union extending over a long period of time. It is the proposition of putting in a transplant. We will be able to do that with little trouble because there is no displacement of the fragments. We must remove that excessive exostosis from the lower fragment.

The mass on the upper end of the lower fragment is the callus. It is a little surprising that there has not been a bony union when such a large callus had formed on one of the fragments. There is one unpleasant element about this case, and that is the large amount of scar tissue in the midline of the leg which I must remove, so that I may be able to get a primary union of the soft parts after the operation. If one does not get a primary union of the soft parts after operation, the transplant resembles a sequestrum, and must be removed subsequently because it becomes an infected dead fragment of bone.

I will make the incision in the line of the scar, excising all the scar tissue. That gives us a good exposure of the fracture. That large callus is on the upper end of the lower fragment. The pseudarthrosis and the line of non-union show up nicely. The callus is the continuation of the lower fragment trying to splint across the gap. Fibrous tissue is interposed between the fragments and is the probable etiologic factor of the non-union. I am taking out that fibrous tissue, as it would increase in amount and become cartilaginous, which is essential in the production of a joint, but has no advantage in ossification. There was no bony contact of the fragments. We will clean that out thoroughly and freshen the ends of both fragments. We chisel out all this callus so as to square off the ends of the fragments. This callus is not hard. It is brittle because of imperfect osteogenesis. It represented an effort on the part of nature to produce a bony union, but with all this cicatricial fibrous tissue between the ends of the fragments union never could take place. It is a typical pseudarthrosis.

We will ream out the medullary canal in both fragments so as to fit the transplant which we wish to insert. Then we will remove an oblong block of bone, one-third of an inch wide and one inch in length, from the anterior surface of the end of each fragment, making a groove into which the transplant can be slipped on its insertion into the medullary canal. It is not neces-

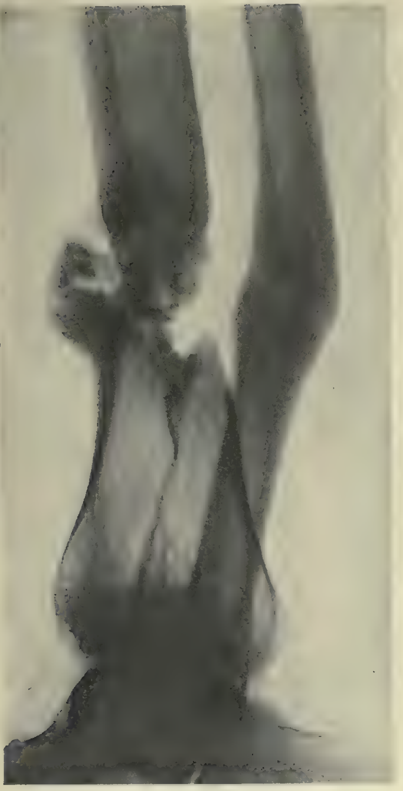

Fig. 169.—Ununited fracture of the tibia (lateral view). The fibula fragments are firmly united by osseous union, although at an obtuse angle, the angulation being backward. The tibia fragments have not united in spite of the excessive production of callus on the ends of both fragments, especially the lower. This lower callus extends up on the side of the upper fragment.

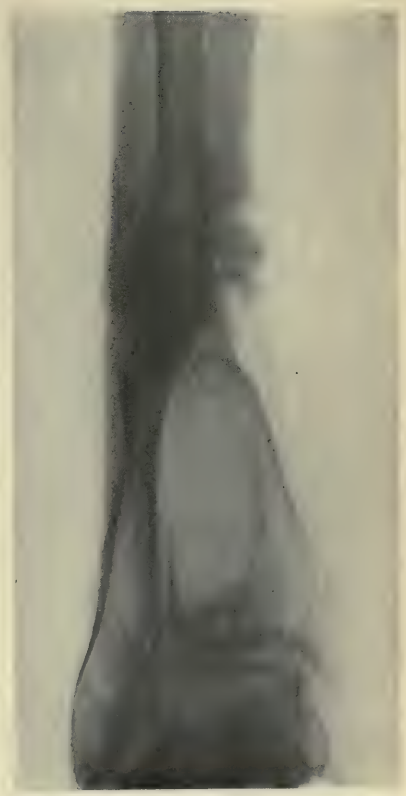

Fig. 170.—Ununited fracture of the tibia (anteroposterior view). The fibula appears to be in good position in this picture, although it overlaps the tibia. The angulation of the fibula seen in the lateral view does not show here.

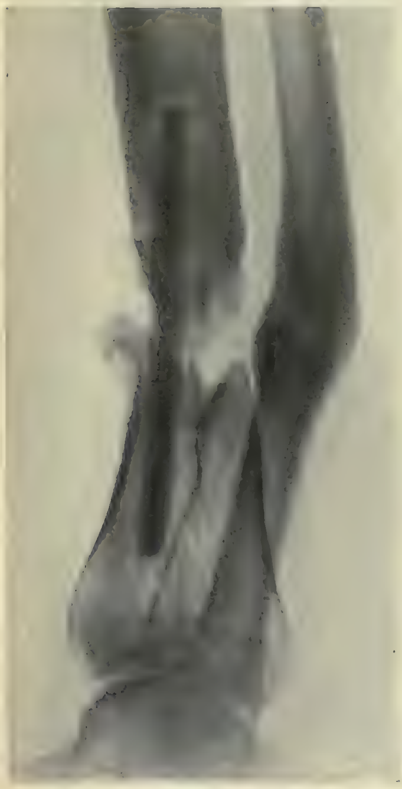

Fig. 171.—Ununited fracture of the tibia. Lateral view, made about six weeks after the operation. The transplant shows very distinctly.

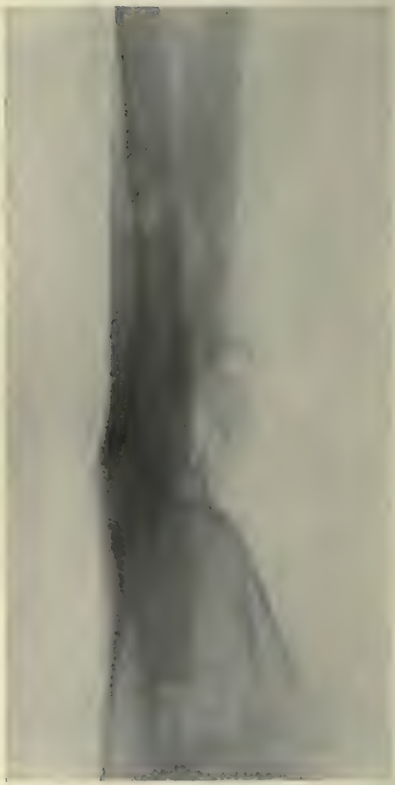

Fig. 172.—Ununited fracture of the tibia. Anteroposterior view, made about six weeks after the operation. The transplant does not show very well because of the overlapping of the fibula. The lower end of the transplant is clearly visible.

sary to make that groove or trough, but it simplifies the insertion of the transplant materially.

This leg will now be covered with sterile towels, while we remove the transplant from the crest of the opposite tibia, which was previously prepared for operation. We make the incision just to the outer side of the crest of the tibia, dividing the aponeurosis of the tibialis anticus and retracting this muscle so as to expose the bone. We remove the transplant from the crest of the tibia with the Zapffe motor saw.

I will remove the periosteum with the transplant. See that saw is just about the right diameter to give me the depth I desire! It is a small circular cross-cut saw, one inch in diameter. That saw is a very nice instrument. It saves very much time and energy, and it is more exact than the chisel. We take out a piece 4 inches in length by ½ by ⅜ x ⅜. The defect in this tibia will fill in rapidly with bone, as we have been able to demonstrate in two cases in which a second transplant had to be removed because of another fracture. The muscle aponeurosis is approximated with catgut and the skin-incision with horsehair. The wound will be sealed with collodion gauze.

The transplant will now be placed into the other leg. I insert the transplant into the enlarged medullary canal of the upper fragment. Then I will drive it up into the upper fragment in place of using a nail to fix it as we sometimes do. Then we slide it into the groove in the lower fragment and drive it down into the medullary canal. I just catch it on the side with the chisel and gently drive it down. It cannot get out. It does not need a nail to hold it.

That completes the operation, except for the closure of the wound. The first step is to put in a subcuticular catgut suture to relieve the skin of all tension, to obliterate all dead spaces between the skin and the transplant, and it gives us a covering for the bone that is an additional support for the transplant and it keeps it secure. The skin-edges are approximated with horsehair, and after dusting with bismuth subiodid the wound is sealed with collodion gauze. A full plaster-of-Paris cast is applied, extending from the toes to the groin. It is split with the Gigli saw as soon as it is hard, and a window is cut out over the head of the fibula, to obviate pressure on the external popliteal nerve.

[The stitches were removed on the sixteenth day. Primary union. Patient up on crutches in five weeks after operation. Plaster cast replaced by long leather cast, reinforced by long steel bands a week later.]

LAMINECTOMY FOR ANEURYSMAL SARCOMA

Discussion of Neurologic Phase of Case By Dr. D'Orsay Hecht

Dr. Murphy: Dr. Hecht, will you please take up the history of the case from the neurologic standpoint?

HISTORY

Dr. Hecht: Agreeable to the wish of Dr. Murphy, I shall attempt a résumé of the symptoms and clinical findings since first they made their appearance in this patient some eight weeks ago. I feel that it would be idle to speculate as to the earliest beginnings of the underlying insidious pathologic process; on the other hand, the extremely rapid accession of clinical manifestations to the point of total paraplegia in so short an interval as two months should receive proper emphasis.

Mr. N., whose family history is negative, is a young man thirty-six years of age, married, and the father of two healthy children. A Neisserian infection acquired at the age of twenty-one had for its more or less immediate sequel an arthritis of the right knee-joint of some five months' duration before complete recovery took place. At odd times, too, during the past four or five years, there have been acute exacerbations of a chronic mucous colitis which have, on the whole, yielded rather readily to appropriate treatment. One year ago overwork and the cares of a new and growing business enterprise caused the patient to feel generally "run down," but under a rest régime the debility soon disappeared. I should add that syphilis is stoutly denied. A negative Wassermann and the absence of somatic criteria for lues tend to sustain this denial. The personal habits of the patient have always been exemplary. There is no history of trauma that can have a bearing on the present pathology. The first physical examination revealed a small circumscribed area about the size of a silver quarter, just to the left of the body of the sixth dorsal vertebra, which was painful to deep thumb pressure, but on close inquiry it was found that the patient has been conscious of this tender spot for the past year or so.

Now, as concerns the present illness: It began with pains, which may be characterized as of the root type, and obstinate gastro-intestinal symptoms. At first the pain was confined to the dorsolumbar region, of a dull, aching, boring, rather than lancinating character, not constant, and not particularly aggravated by motion or walking. The greatest distress was felt at night on efforts at lying down. A half-recumbent position, although uncomfortable, was at all times preferred to lying prone in bed. In fact, the latter position has for the most part been impossible. Efforts at straightening up in an erect position have from the first been associated with a feeling of tension, and what the Germans aptly call "Spannungsgefühl," in the left groin and hypochondriac region. Contrary to type, the pains have never shown a tendency to radiate into the sides, after the fashion of an intercostal neuralgia, or toward the front of the chest or abdomen. Nor have they shown a disposition to descend into the thighs or legs. They have remained away for days at a time, but when present, have been almost unendurable. About four weeks after their onset the patient experienced the first typical girdle sensation. If my comment of the element of pain is extended, it is because the variability of radicular pain is misleading, and too often creates an impression in the mind of the practitioner of a simple, innocent neuralgia and is too readily dismissed as such. I for one am prone to frown on all back pains, especially when they are neuralgiform, until I can satisfy myself as to the possible underlying pathology.

I would next call your attention to the intestinal distress. Almost from the first the chief complaint was one of a bloated feeling, uninfluenced by the kind or amount of food eaten. There was much belching, and although flatus was passed in large amount, it brought no relief from the distressing sense of

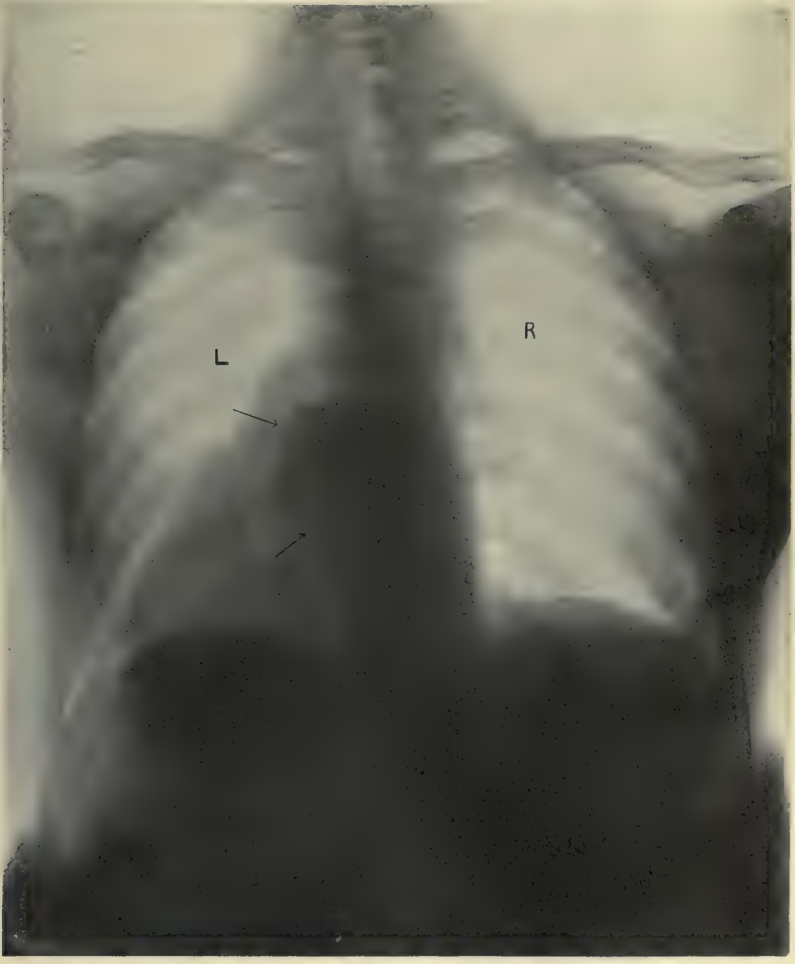

Fig. 173.—Aneurysmal sarcoma: A part of the tumor shows clearly to the left of the spine, extending from the seventh to the ninth rib as a darker shadow, also overlapping the spine. It is within the heart shadow.

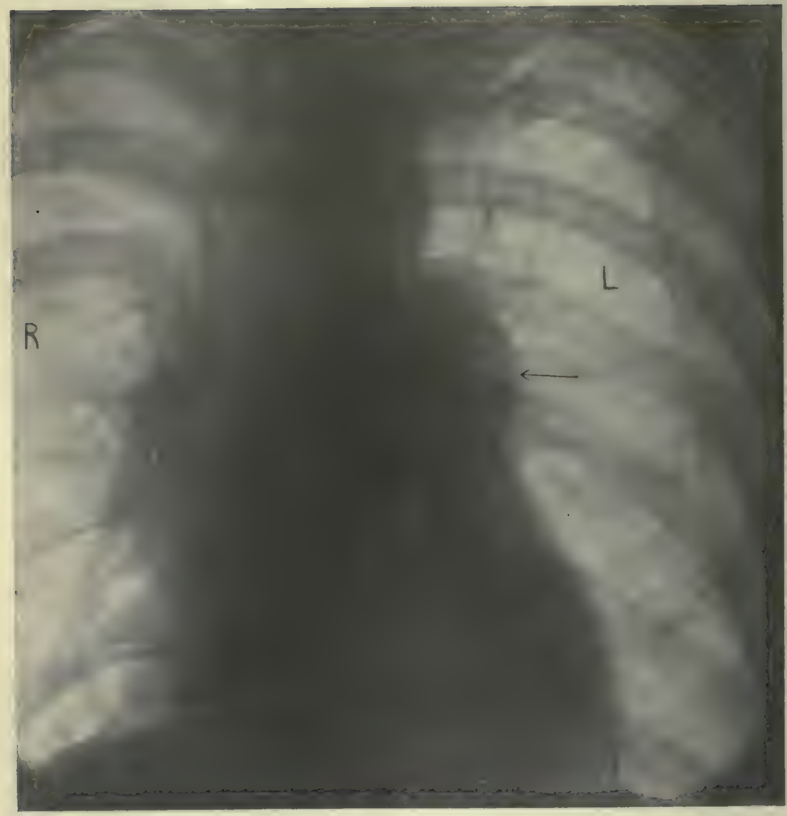

Fig. 174.—Aneurysmal sarcoma seen from in front. The tumor shadow is to the right of the spine and heart shadows.

fulness and distention in the transverse and descending colons. The diet, oil enemas, and other therapeutic measures, given with good effect for previous attacks of mucous colitis, failed to allay the bowel condition in the present emergency. In view of the increasing back pain and refractory visceral symptoms, the patient was admitted to the Michael Reese Hospital for more extended observation. The gastro-intestinal manifestations received the close attention of my colleague, Dr. Walter Hamburger, who previously, on occasion, had seen the patient with me for digestive disorders. Inspection revealed a well-defined dorsal scoliosis to the right; undue prominence of the right scapula and a lowering of the left shoulder of two inches, as compared with that of the right. This deformity had been so noticeable during all the years that I had known the patient that *per se* it assumed no special importance at this time. The erector spinæ on the left were markedly rigid and prominent. Percussion of the chest posteriorly gave a well-demarcated strip of dullness, even flatness, about three fingers in width, just to the left of the bodies of the sixth and seventh dorsal vertebræ. With this finding the thought of an aneurysm was kept well in mind, but careful auscultation of the heart over the large arterial trunks proved negative. Examination of the abdomen with the patient in a prone position gave an absence of visible peristalsis. The left upper abdominal quadrant was conspicuously distended and the left rectus abdominis distinctly rigid. In fact, the entire left side of the abdomen felt tense and tympanitic and remained so most of the time; flexion of the limbs or change of position otherwise seemed not to affect it.

On the part of the central nervous system, the pupils and cranial nervous were intact. The tendon-reflexes were everywhere present, equal, and normally brisk. Babinski sign was negative. The abdominal skin responses were normal, and Beevor's sign, a criterion of focal importance, pointing to a lesion involving the tenth dorsal segment, was absent. Gross muscular power and strength were unimpaired, and sensory disturbances there were none. The sphincters remained normal.

This early hospital observation included the taking of the two skiagraphs. There is a dense shadow, smooth and regular in outline, occupying an area that corresponds with the dulness already referred to. The vertebral outlines are well preserved, and the shadow is denser even than that of the adjacent column of bone. In one of the skiagrams the tube was to the front, the plate to the back, and the patient lying down. In the other skiagram the mass shows with the conditions exactly reversed, viz., the patient sitting erect, the tube to the back and the plate in front. (See Figs. 173 and 174.) Fluoroscopic examinations undertaken several times to determine the relation of the mass to the aorta were not satisfactory.

For purposes of completeness, permit me to call your attention to the laboratory data: Several examinations of stomach-contents and feces were negative. The sputum was negative. The twenty-four-hour urine showed more than a trace of albumin and a few hyaline casts. The blood-counts were negative, except for the appearance of five eosinophiles in a field of 100 cells. The blood-pressure was 110. As I have already indicated, two Wassermann tests were negative. The spinal fluid was not drawn because of the increased discomfort and irritability it would have caused the patient. As you may well imagine, the repeated analyses and examinations did not have a tranquilizing effect on the patient; he grew restless and apprehensive from loss of sleep and pain, and left the hospital to return to his home.

We may, I think, at this point, summarize the positive findings of record thus: Root pains associated with indefinite, though suggestive, visceral symptoms; the presence of a mass determined by percussion and evident in the skiagrams; also erector spinæ rigidity and dorsal scoliosis. Taken together, they are significant of impending spinal cord disease, but the total absence of focal symptoms would leave one much in doubt as to the degree of involvement or the kind of intervention best suited to the condition.

Within the next two weeks the clinical picture was clouded by the appearance of many symptoms of a functional character, induced by much introspection, anxiety, pain, loss of sleep, and general irritability. After a trial of rest at home, during which the mental irritability of the patient seemed on the increase and the objective findings remained unchanged, a series of mud-baths were advised, chiefly with a view to controlling the agonizing pain. The experience at these baths, taken away from home, was limited to only a few treatments, and cut short by the increasing discomfort.

The patient was now approaching the fourth week of his disability, when he complained for the first time of a sense of numbness in both feet. Regarding this subjective sensory disturbance as the forerunner of other focal manifestations, I subjected Mr. N. to another rather prolonged, and for him tedious, examination. I was able to satisfy myself for the first time that there was a slight extensor weakness in the left leg; a reduction of the abdominal skin reflex on the left; a slight disparity between the tendon-reflexes in both the arms and legs; a difference in the cremasters and an indication of left patellar clonus. A painstaking sensory examination proved negative. I, at that time, gave it as my opinion that the first definite compression symptoms were at hand. I should not forget to mention that just prior to this examination the patient had been subjected to a series of important tests to determine the origin of all possible foci of infection that could have led to the painful back symptoms. For instance, a complement-fixation test for gonorrhea was made because of the history of a gonorrheal arthritis. The tonsil was also looked into for evidence of a submerged pus-pocket from which resorption might take place and thus lead to systemic infection; likewise the prostate was examined and milked for evidence of retained infection and to ascertain primary malignancy in anticipation of the tumor mass in the back being metastatic in character. From this you may infer that prior to the development of compression symptoms every effort was made to clarify the possible inter-relation between a primary focus and secondary invasion from infection or neoplasm. From the tenth of April on the paresthesias in the feet began to ascend to the knees and thighs, so that by the time of operation they had reached to about the umbilicus. With this exception, the findings again remained practically stationary until

April 27, 1913, when the weakness in the legs amounted to a definite paraparesis. There was a reduction of the tactile sense in the legs extending to the knees, and at times an inability to recognize the position of his feet in space; in short, a disturbance of the muscle sense.

On April 28th the paraparesis developed into an almost complete paraplegia; the Babinski sign appeared on the left; the tendon-reflexes everywhere on the left were now markedly increased; the thighs assumed an abducted position, with the feet slightly everted; the hypesthesia was changed into an anesthesia, extending almost up to the costal arches; vesical retention appeared for the first time. The rapid progress to a complete paraplegia with sphincter involvement, as you see, required but a few days. The first localizing signs preceded this complete paraplegia by twenty days.

You may ask why prompt intervention in the form of a spinal decompression was not advised at the very onset of compression symptoms. That query is perfectly proper, and can be met only by the contention that the nature of the lesion, in the sense that it might be an aneurysm, was a forbidding one. The very rapid course of the disease, the significant density of the tumor mass, a density even greater than that of bone, together with its position, narrowed the clinical diagnosis down to practically just two possibilities, namely: an aneurysm or an osteosarcoma of the spine.

The diagnosis of an aneurysm of the descending arch of the aorta or a mediastinal tumor must always be borne in mind when we are confronted with root-symptoms in the dorsal region, either associated with or without evidences of paralysis. Assuredly a diagnosis of an aortic aneurysm is easy in the presence of specific physical findings, such as the bruits, the differential pulse, etc., but aneurysmal dilatations can be present without conspicuous cardiovascular findings, and it is in these very cases where the refractory intercostal neuralgias and other spinal symptoms may be the sole criteria of aneurysm that error, though pardonable, must be guarded against. Again, compression myelitis symptoms having their inception with spinal root pains should never fail to conjure up the possibility, and not a remote one either, of vertebral tumor, of which carcinoma and sarcoma are the most frequent of occurrence. In this particular case the idea of a carcinoma could with reasonable certainty be excluded, as it can be in nearly all cases, on the theory that for it to be a carcinoma of the spinal column it must have been preceded by primary malignancy elsewhere. In short, carcinoma of the spinal column is almost never, and I should not hesitate to say never, primary. Thus carcinomata that have been declared primary in the spinal column have, in more recent times, owing to our better interpretation of the histopathology, come to be known as either alveolar sarcomata or endotheliomata, and as such are to be strictly differentiated from carcinoma. It is of clinical value to remember that in metastatic carcinoma of the spine appearing in the male the seat of primary malignancy should be looked for in the prostate. In this case you recall that this was done.

Now we come to the sole remaining diagnostic consideration in this case, namely, sarcoma. Sarcoma occurs as a primary as well as secondary growth in various forms in the spine, and is likely to attack any part of the bony column. In this particular case the very rapid progress would point to a rapidly destroying dissecting form of sarcoma, and, as concerns this conclusion, I find myself in perfect agreement with Dr. Murphy, who will, no doubt, give you the benefit of his surgical attitude as he proceeds with the laminectomy he is about to perform.

It only remains for me to say that a localizing diagnosis from the neurologic findings alone can now be made very definitely, and the surgeon advised to resect the spines that will lay bare the segments from the sixth to and inclusive of the tenth, of the dorsal cord

In this presentation I have no doubt occupied a disproportionate amount of your time and Dr. Murphy's, but it occurs to me by way of apology to add that a case so interesting in its clinical and pathologic aspects should not be summarily dealt with.

COMMENTS AND OPERATION

Dr. Murphy: The presentation of the neurologic phases of the case by Dr. Hecht leaves little to be said from a surgical standpoint. It is a case of almost complete transverse paralysis, occurring speedily, with a history of preceding pain in the nerve-roots. The skiagram shows a tumor, 1½ inches in diameter, clean cut—as clean cut as the aorta—on the right side of the spinal column. His pain extends down into his limbs. There is a severe pressure backward against the arches of the vertebræ from something that is situated anterior to the spinal column—that is, it has come through from in front. This is demonstrable by pressure on the tips of the spinous processes, which causes excruciating pain.

We have gone over the factors that produce an ordinary osteochondroma. An osteoma in the spinal column usually takes its origin in the bodies of the vertebræ. Osteoma in this location is not common, and it rarely ever compresses the cord. When it does do so, the compression occurs very slowly, not producing the superlative symptoms noted in this case before many months or even years. Then one must consider the acute infections in the bone, such as an acute osteomyelitis with a granuloma or an abscess pressing back toward the spine. There has not been any elevation of temperature at any time, and no other evidence of infection in the history of this case. Again, if this mass is an abscess, it would show in the skiagram as an area of rarefaction and not solidification, as is the case now. Echinococcus cysts and actinomycosis also occur in the spine. These, too, would show a rarefaction. This skiagram, however, shows a solid mass, not a rarefied one. We cannot distinguish the right margin of the tumor definitely. What can this be, if this dark shadow is not thrown by blood? A bone lesion compact enough to give a shadow as dense as this one is found only occasionally in the metaphysis of the knee, that is, the slow ossifying processes that take place in the shaft of the long bones as the result of infections that are active over a period of years. Of such this man has no history. There is no history of trauma, no lues, nothing that would cause an aneurysm in that portion of the aorta, as is indicated in the skiagram. Occasionally, an aneurysm in this location causes an absorption of bone and finally compresses the spinal cord. An aneurysmal sarcoma could develop as rapidly as this condition has and compress the cord, but such a lesion should give a rarefaction and not a solidification. An aneurysmal sarcoma should give a skiagraphic picture similar to that of an aneurysm.

We approach this operation "with fear and trembling," because of the bleeding that may occur and possibly lead to a fatal termination on the table, as we interpret this to be an osseous aneurysmal sarcoma. That is one of the conditions we are prepared for, and his family has been prepared for it—that he may bleed to death on the table. We hope to be able to keep clear of the borders of this mass long enough to determine its character, and if it is an aneurysm or an aneurysmal sarcoma, as we believe, we will at once close it up and avoid such a dire calamity as a death on the table.

I think the lesion lies right at the seventh dorsal vertebra. I will make a longitudinal incision over the spinous processes, extending from the fourth to the eighth dorsal vertebræ. There is, you will note, considerable hemorrhage as soon as I cut down on the spine. I will compress that wound until all the bleeding stops. As I retract the periosteum from the right side of the spine, between the sixth and seventh dorsal vertebræ, I see a mass of granulation tissue, and, removing it a little more with my finger, there is a gush of blood. This is undoubtedly an aneurysmal sarcoma between the sixth and the seventh vertebræ. That corresponds with the skiagram, and accounts for the speedy development of the case. It is an aneurysmal sarcomatous destruction of the body of the vertebræ. I block this opening with a sponge while I place the figure-of-8 silkworm-gut sutures deep in the muscles so as to force this thick pad of muscle against the spinous processes, which, fortunately, I did not remove. That will prevent the formation of a hematoma and the occurrence of secondary hemorrhage. We will not attempt any further procedure.

Dr. Hecht: How do you feel about the subsequent course of this case in point of time?

Dr. Murphy: It is very short. He had no elevation of temperature. That is contrary to the rule when they grow as rapidly as this. My first case of this kind was very striking. It occurred when I was an intern in the Cook County Hospital and in the service of the late Dr. Fenger. The patient was a young German who was working as a stevedore. He was carrying some boards when he slipped and fell on his knee. He worked that day and the next day. On the third day he began to have some pain in his knee and he had a high temperature. He came into the hospital on that day with a temperature of 103° F. and with great pain in the knee. If I had that case today I would say it was a case of acute infective osteomyelitis occurring in the metaphysis of the femur. The next day he had a temperature of 104° F. and he was delirious. Dr. Fenger examined him. He did not know just what to make of the case. Finally, about the fifth day, he began to have evidences of bone absorption. Then he felt that it was an aneurysmal sarcoma, and such it proved to be. Fenger operated on him, amputating the leg at the hip. He made a good recovery from the operation. I do not know the subsequent course of the case, as the patient left for Germany immediately on his recovery.

Let the record show that there was a sarcoma protruding between the transverse processes and the laminæ of the sixth and seventh dorsal vertebræ; that it was probably a little below the center point of the elevation; that there was profuse flowing of blood, as from an artery; that this was controlled by pressure. We did not remove any of the laminæ or spinous processes. If I took them off, I would have had much less hope of control of the hemorrhage than I had. We did not make any effort to determine the extent of the destruction of tissue on account of the bleeding. There was a mass of granulation tissue presenting, as long as the lower two segments of my third finger, showing that it was the so-called aneurysmal type of sarcoma. That shows that we were correct in the diagnosis. That is good for that class of case, because these cases are so rare. It worked out better on

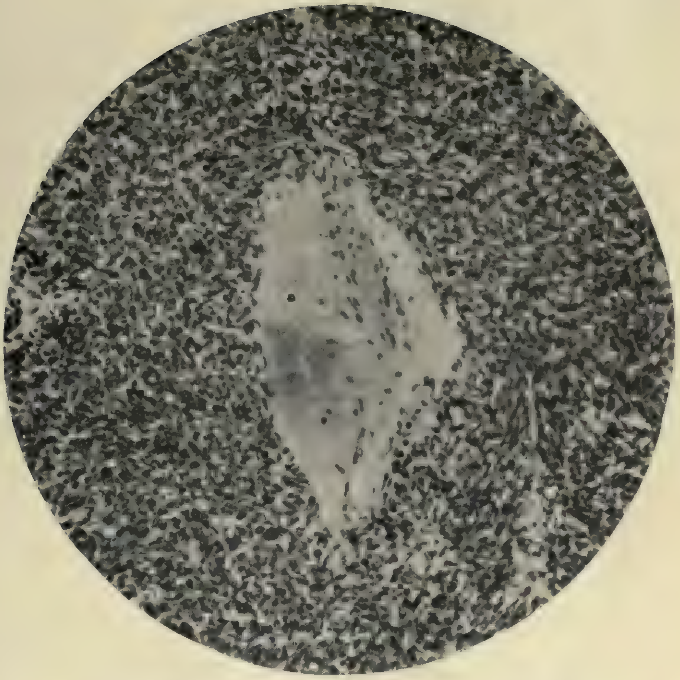

Fig. 175.—Photomicrograph of section showing small round- and small spindle-cell sarcoma traversed by blood-vessel without wall. Mixed-cell sarcoma of spine.

the table than I thought it would. I told his people frankly that I believed he would die on the table.

[A microscopic section made of a spoonful of tissue removed at the time of operation confirmed the clinical diagnosis of aneurysmal sarcoma. There was a primary wound healing. He had a trophic sloughing over the sacrum and heels. He has emaciated greatly, and is now only a living skeleton. There has been no enlargement backward of the tumor. The last skiagram, made thirteen weeks after the operation, shows no material change in the size of the tumor. He has been getting the Coley serum and Röntgen-ray treatment, but nothing seems to retard the progress of the disease. The fatal termination is close at hand.]

EXPLORATORY LAPAROTOMY IN A CASE OF SEVERE ASCITES IN A GIRL FIFTEEN YEARS OF AGE

WITH REMARKS BY DR. PAUL CHESTER

Dr. Murphy: Dr. Chester, will you kindly take up the history of this case and the analysis of it? Dr. Chester has had the case referred to us from his department.

HISTORY

Dr. Paul Chester: This patient is fifteen years of age. The history dates back to eight months ago, August, 1912, when a swelling of the face, extremities, and abdomen began. This was slight in extent until last January, when the swelling of the abdomen increased very rapidly. At the present time the patient has an enormous distention of the abdomen. This distention is caused by fluid in the peritoneal cavity—an ascites. This fluid, of which we drew off 2½ gallons at one sitting, is a transudate and has a specific gravity of from 1005 to 1009; it contains a few white and red blood-cells, and possibly 0.5 per cent. of albumin. Now we propose to make a clinical diagnosis in two ways: first, by exclusion, and, second, by an exploratory operation.

The causes of ascites may be found in diseases of the lungs, heart, liver, kidneys, pancreas, ovaries, peritoneum, blood, and portal vein. Emphysema of the lungs with secondary dilatation of the heart may give rise to ascites as part of a general dropsy.

In the heart we find that myocardial weakness, valvular disease, and adherent pericardium may act as causes of ascites. Cirrhosis of the liver, syphilis, and cancer commonly cause ascites by obstructing the portal circulation.

Nephritis, when accompanied by ascites, is usually associated with cardiovascular disease, so that the local dropsy is part of a general edema due to one or both of these conditions. Pancreatitis and cancer of the pancreas and glands about the portal vein may account for ascites in some cases.

Diseases of the peritoneum, especially tuberculous peritonitis and malignant disease of the abdominal organs, with extension into the peritoneum, producing malignant peritonitis, are always accompanied by ascites. There is also an essential hydrops peritonæi. Pernicious anemia and leukemia are sometimes causes of ascites. Finally, diseases of the portal vein, causing direct obstruction, such as thrombosis of the vein or the pressure of tumors or peritoneal adhesions, result in ascites.

Now, in this patient we can exclude diseases of the heart and lungs, because they are apparently normal. In the case of the liver we find much more difficulty because we notice that the usual alcoholic history pointing to cirrhosis is absent; also we find great difficulty in determining the size and consistence, condition of the surface and edge, because of the fluid present which obscures the physical signs. When we drew off the fluid we found the liver apparently decreased in size, since it is not readily palpable, except that a mass could be felt under the costal arch to the right side.

We have yet to consider the possibility of cirrhosis, syphilis, and cancer of the liver. The age of the patient—fifteen years—speaks against cancer and cirrhosis, as does the absence of alcoholic history, which I mentioned before. Congenital syphilis, which might be present at this age, can be excluded because of the lack of other syphilitic findings and the fact that the liver is usually enlarged in syphilis, and also the Wassermann reaction is negative.

The fact that the fluid present is a transudate shows that there is an obstruction of the portal circulation, either in the vein outside the liver or within the liver. If we can definitely exclude syphilis and cancer, we then have cirrhosis remaining as a possible cause of the ascites.

That the portal vein itself is involved is suggested by the rapid return of the dropsy after tapping, the distention reaching its original size within twenty-four hours.

By examination of the urine we find a trace of albumin constantly present, together with hyaline casts. These findings in the urine led at first to the diagnosis of nephritis. Now I believe we can exclude nephritis as a cause of the ascites because the amount of albumin remains practically the same all the time, the casts are not always present, uremic symptoms are absent, and the distribution of dropsy is atypical for nephritis. It may be assumed that the presence of albumin in the urine is due to pressure of the ascitic fluid on the renal vessels, and not to any damage to the kidneys themselves.

We can exclude tuberculosis and malignant disease of the abdominal organs involving the peritoneum because the fluid present is a transudate, and not an exudate, as it would be in either of these conditions; also the tuberculin reaction is negative. The blood is normal.

Therefore we may conclude that the ascites is produced by obstruction of the portal vein, either within or without the liver, and that the obstruction is practically complete.

VISITING DOCTOR: May I ask you to explain the difference between an exudate and a transudate?

DR. CHESTER: A transudate is a fluid that has escaped from the blood-vessels as a result of increased pressure in the veins. A transudate has a low specific gravity—less than 1015. An exudate is the result of inflammation or irritation, and has a specific gravity higher than a transudate. If the specific gravity is above 1018, it is always an exudate. Of course, an exudate contains other elements showing inflammation,—bacteria, fibrin, blood-cells, epithelial cells,—while a transudate is always clear fluid with a low specific gravity containing but few cells.

COMMENTS AND OPERATION

DR. MURPHY: There are five conditions which produce fluid in the peritoneal cavity. They are: First, diseases of the kidney; second, diseases of the heart; third, diseases of the lungs; fourth, diseases of the liver, and fifth, constitutional or blood diseases—in addition to the lesions that occur in the peritoneum. Any one of these may cause a transudate into the peritoneal cavity. They lead not only to ascites, but also to effusion into the cellular tissue, causing edema of the face, hands, and extremities. Dr. Chester considered the other class of lesions that would tend to cause an effusion into the peritoneal cavity as a primary consideration. Obstructions to the portal vein, associated with lesions of the liver and of the peritoneum, usually give rise to ascites of varying degree. Most common is that associated with tuberculosis; second, the hydrops type, in which there is an exudate from the peritoneum—a hydrops peritonæi. The fluid in that case does not contain blood, and there is no demonstrable lesion except a thickening of the peritoneum, the same lesion as in a hydrops of the knee, due to a thickening of the synovial membrane.

Then he mentioned lesions of the pancreas causing a hydrops associated with a fat necrosis, and leading to an obstruction of the portal circulation, thus producing a stasis and a transudation into the peritoneal cavity. Then we must also bear in mind the lesions that result from obstructions of the chyle-ducts, where the abdomen is full of a chylous fluid that is easily recognizable because of its milky appearance.

Next are the malignant lesions that occur in the peritoneal cavity. The most common of these is sarcoma. A sarcoma generally produces an exudate in large quantities. Then there is also carcinoma, this being the next most common malignant lesion in the peritoneal cavity. Both of them, if they involve the peritoneum, frequently cause a bloody exudate, and this is very suggestive, if not pathognomonic, of malignant disease, except for rupture of the tubes and other organs. Lesions in the female genitalia also may be responsible for an exudate in the peritoneal cavity, such as—(*a*) Malignant lesions of the ovary, ovarian cysts, sarcomata, and ruptured ovarian cysts. (*b*) The type of villous growths, the papillomata, that occur in the peritoneal cavity, is the lesion that causes an exudate great in quantity. A mucoid or mucilaginous material is sometimes found in the peritoneal cavity as the result of a ruptured cyst. The secretion from the wall of the cyst is a mucoid material which is not absorbable. It is often found in great quantities in the peritoneal cavity. Then there are the cysts of the urachus and echinococcus cysts of the liver that rupture into the peritoneal cavity, and cysts of the gall-bladder filling the peritoneal cavity with large quantities of fluid, occasionally mucoid in character, with or without the presence of gall-stones, or a large quantity of bile may be poured into the peritoneal cavity. Cirrhosis of the liver, the sequence of a pericarditis, is one of the lesions that should be seriously suspected in these cases, but this patient has no history of pericarditis and shows no evidence of pericardial synechiæ.

Dr. Chester has gone over the whole subject of effusions into the peritoneal cavity. Then he endeavored to make his diagnosis by exclusion, throwing out first one then the other cause, until he came to the final conclusion that this is a transudate and not an exudate. Therefore it is not one of the inflammatory conditions that occur in the peritoneum or its neighborhood which cause a constant pouring-out of fluid into the peritoneal cavity. From the age of the patient (fifteen), ordinary causes of obstruction to the venous circulation, such as a cirrhosis of the liver, are not to be considered. Whether she has a disease of the liver I do not know, although the liver appears to be slightly enlarged and hard.

She presents evidences of an extensive obstruction. The ascites is of severe degree. There is an enormous distention of the abdomen. We will not endeavor to do an anastomosis of the portal vein, but we will decide on some method of side-tracking this effusion, if possible. We may transplant a portion of the omentum into the cellular tissue, so as to drain the fluid out in that way. Or we will drain the fluid into the cellular tissue of the subclavicular region by means of several strands of tubular silk (Handley).

(Operation April 17, 1913.) We will make the incision as for an exposure of the gall-bladder, going through the right rectus muscle. There is no fixation of the omentum. See how quickly the fluid comes out! It is a perfectly clear fluid, as described so well by Dr. Chester, showing you why he called it a transudate and not an exudate. It has a low specific gravity, and contains practically no albumin. The fluid has been evacuated now and we can inspect the liver and the portal vein. It is a lesion of the liver. The liver is contracted down into a small mass with a dilated gall-bladder. It is studded with neoplasms, ranging in size from a pea to a hickory-nut. This liver is not more than 5½ or 6 inches in width, and it is only as thick as my hand. It is not adherent to the diaphragm. I am able to displace it easily and bring it into view. It looks more like a cirrhosis of the pancreas. The hepatic artery is in view. The portal vein is embedded in a mass of induration. The masses are under the capsule of the liver, and not outside of it. I believe that it is either a specific or a malignant lesion. If it is a malignant lesion, it is probably an adenocarcinoma, although that is inconsistent with the atrophy. It may be simply a multilobular atrophic cirrhosis.

There is no evidence of enlargement of the lymph-nodes below —nothing to indicate that this enlargement of the liver is secondary to the enlargement of the nodes. With this condition present there is no reason for attempting to implant a portion of the omentum. We will put the ends of tubular silk into the abdomen and carry the strands up under the skin as high as the clavicle. That should carry this fluid rather rapidly into the cellular tissue. That silk leads from the peritoneal cavity out under the skin.

Now I will close the peritoneal cavity and the incision in the usual manner.

SUBSEQUENT HISTORY BY DR. CHESTER

The patient died three days after the operation, and as only a limited postmortem examination was permitted, the liver was removed and examined by Dr. F. Robert Zeit, pathologist of the Hospital, who reports that the pathologic diagnosis is atrophic cirrhosis of the multilobular type.

This case is of great interest because of the age of the patient, and also because of the absence of alcoholic history. Cirrhosis of the liver in young persons is so rare that it was not given sufficient consideration in the study of the case.

In looking over the literature we find that a number of cases have been reported in young children—one as young as sixteen months. In some of the patients no history of the use of alcohol could be elicited, while in others careful inquiry showed that even in these young subjects a definite history of beer or wine drinking could be obtained. These cases without alcoholic history show that the typical pathology of Laënnec's cirrhosis may be produced when alcohol is not an etiologic factor, and that other sources of irritation of the liver must be looked for in these patients.

It is easy to understand that various toxic substances might be introduced into the intestines or be formed there, and by continued irritation of the liver produce cirrhosis. Ascending infections may also lead to similar conditions.

Dr. Murphy: It is very unsatisfactory when a case of this kind comes to us, because even after careful clinical observation and postmortem examination we have not learned to make a diagnosis of a similar condition in another patient. We have not learned the etiology of the disease, and know nothing more of the treatment than we knew before. We have only learned that such a disease is possible; that it does occur; that it must be reckoned with in clinical work.

VESICAL CALCULUS WITH A HISTORY SIMULATING PROSTATIC DISEASE

HISTORY

Patient, male, aged fifty-nine, comes to hospital on account of pain in the suprapubic region. The trouble began a year ago (April, 1912). Since then the patient has had to urinate seven or eight times during the day and three or four times during the night, passing only a small amount of urine at a time. At times the stream starts easily; at other times very slowly. Then, again, it is interrupted. Last summer he passed blood mixed with urine after he had exerted himself by walking. This happened on four or five occasions. The pain is constant and worse just before urinating. Often, before urinating, he has pain in the back on both the right and the left side. Past history negative; venereal, negative.

Examination showed only a moderately enlarged prostate, not sufficient to account for the urinary obstruction symptoms, but size is only one of the elements of urinary obstruction from the prostate.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 25, 1913): This man is one of those fat, puffy fellows with a varicose condition of the veins of the bladder. The prostate is only moderately enlarged. He had a cystitis at one time, which has subsided. We have had some cases of varicosities of the bladder that resembled angiomata in size of the blood-vessels. There is considerable bleeding in these cases. In the ordinary case of removal of the prostate there is comparatively little hemorrhage, but these cases bleed a great deal. Occasionally we have a hemorrhoidal dilatation of the blood-vessels in the bladder-wall. These may ulcerate and bleed profusely, or rupture, as they do in rectal hemorrhoids. We never have to remove the prostate on account of the presence of the varicose veins, but we will first examine the bladder carefully because he may have a vesical calculus, although he has no symptoms of stone. In cases with a comparatively firm fibroid prostate the bleeding is very slight, but with a fibroid that is not firm the hemorrhage is considerable.

We prefer the suprapubic operation in these cases. We pass a urethral sound first to serve as a guide, keeping the point of the sound pressed well upward. We make a longitudinal incision in the midline, about three inches in length. I am going to divide the attachment of the rectus muscle at each side of the symphysis, so that I can get into the space of Retzius. I am enlarging this opening to give me enough space to get down into this fatty pelvis. There is the great mass of fatty tissue that you always see in this class of cases anterior to the bladder. Look at that fat! It is just like blubber. The urethral sound anchors the bladder nicely. I would not be comfortable in this pelvis unless I knew that the bladder was securely steadied by the instrument. I can feel the tip of the sound through the bladder-wall. Now I will put two silk anchor stitches into the wall of the bladder on each side of the tip of the sound before opening it. We always insert them in this class of cases, where the bladder is situated deep in the pelvis. They make the bladder secure so that it cannot get away from you while you work. That stitch goes into the bladder and out of it. Now I incise the bladder between these two anchor stitches. There is the tip of the sound protruding from the bladder.

I am in the bladder. This man has never been sounded; nothing has been done to him. We never sound these patients before operation. We do nothing that might carry infection into the bladder or traumatize the bladder or urethra in the presence of infections that are so commonly found. Instrumentation has become a pernicious and even dangerous practice in all ancient cases of cystitis due to prostatic obstruction and other factors. Even when it is done carefully, it is dangerous; when it is done crudely, it is wicked. Here is a stone. This stone acted as a trap-door, obstructing the outflow of the urine and caused much of the trouble. The skiagram did not show it. See the size of it and still the *x*-ray did not show it! It is as large as a goose-egg. I am not going to take out that prostate. I am packing this bladder so as to expose the neck where I wish to divide that prostate with the cautery. See the white mass below the sound—a pedunculated, so-called middle lobe of the prostate. That is a beautiful specimen. I do not often get a chance to show one like that. This mass folds right over the urethra. That is what did the mischief. We will remove it with the actual cautery.

Let the record show that there was a tip-like process of the prostate projecting onto and obstructing the posterior urethra; that it was cauterized; that there was a stone about 1½ inches in thickness and two inches long resting in the bladder; that a small drainage-tube was placed in the space of Retzius, and fixed to the skin with a silkworm-gut suture. The bladder was closed with catgut sutures down to, but not including, the mucosa.

Formerly one of the greatest difficulties we used to have was working in a bladder that was septic. Those who went through that in the early days know what it meant to sound a septic bladder. We no longer sound any of these cases.

[The patient progressed rapidly to a complete recovery. The drainage-tube was removed in a week. The wound was closed in three weeks, and the patient left the hospital in four weeks, entirely well.—Ed.]

LAMINECTOMY FOR MYELOMA OF CORD

WITH REMARKS BY DR. MIX

HISTORY

Patient, male, aged forty, enters hospital on account of pain and tenderness over the back in the region of the last lumbar and first dorsal vertebræ. There is a deformity, a backward angulation, opposite the last lumbar and first dorsal vertebræ.

About the tenth or fifteenth of January, 1913, he had a chill lasting one hour. He does not know whether he had any fever. He denies trauma. About February, 1, 1913, he had sharp, stinging pain in the lumbar region. This pain was worse on getting up in the morning. He did not have any pain when lying perfectly quiet. He worked three weeks, when he began to get so weak that he had to stop working. When he entered the hospital about a week ago (April 12th) he was unable to walk, although he could stand. He has had impending paraplegia since the first of April. No knee-jerks, no cremasteric, no Babinski. He has control of bladder and rectum. No girdle sensations. Has to urinate two or three times at night. He had cramp-like pains in the lower extremities at time of admission. He is constipated, has a cough, which he says is due to coal-dust; no jaundice, nausea, vomiting, pain, or tenderness in abdomen. Feeling of malaise. Has lost weight. Previous history negative. Denies venereal disease. Family history negative as to tuberculosis and carcinoma.

COMMENTS AND OPERATION

Dr. Murphy: That history has no value in making a diagnosis in this case except as a gross lesion. I asked Dr. Mix to examine the patient. The findings as we interpret them indicate that the lesion involves the tip of the conus, not the spinal cord itself. He has a complete paralysis—an absence of the Babinski and of all the reflexes. If the lesion were higher up in the cord, the reflexes below the seat of involvement would be intact. Remember that these reflexes do not come from the brain. They are transmitted through the efferent nerve to the center in the cord and come back through the afferent nerve. When he entered the hospital he could stand. Soon after, however,—within nine days,—he was unable to stand or move his limbs at all. Now he has a complete paraplegia. Since the onset of the paralysis he has had a slight elevation of temperature. He has a slight kyphosis in the dorsolumbar region. We made the tuberculin test, but one cannot charge the rise in temperature that followed the injection to the tuberculin, because he has been having a slight temperature elevation right along. If he had a normal temperature and we gave him the tuberculin and elevation followed, then we would say he has tuberculosis, but we cannot say it is tuberculosis with the temperature-curve after the injection so closely resembling his present temperature-curve. If there has been a temperature elevation regularly in the afternoon preceding the test injection, and it does not change materially after the tuberculin injection, then it must be considered a negative reaction. The temperature is coming down now. Whether it is an osteomyelitis or an infection in the cord we do not know.

This is what we must do: We must relieve the cord from its pressure, otherwise its axons are going to be destroyed. Therefore the operation we are going to do will be a laminectomy with a decompression of the cord, even if we cannot remove the cause of the pressure.

He has a pronounced deformity of the spine in the dorsolumbar region. It is very tender to pressure. I do not know whether it is a simple tuberculosis or a tuberculosis with a mixed infection. Deformity and sensitiveness occur in osteomyelitis much more commonly than in tuberculosis.

(Operation April 21, 1913.) We will make a longitudinal incision over the spinous processes, extending from the tenth dorsal to the third lumbar spine. The blood seems to be rather thin and pale. What is the blood-count, doctor?

INTERN: White cells, 10,400; red cells, 4,160,000; hemoglobin, 78 per cent.; blood-pressure, 114.

DR. MURPHY: We retract the tissues to either side of the spinous processes so as to get a good exposure, and then amputate the spinous processes on a level with the laminæ with a heavy rectangular bone-cutting forceps. The body of the second lumbar vertebra is dislocated forward, and therefore the cord is angulated here. This vertebra is loose; it moves back and forth when I take hold of it, which is just the opposite to what it usually does, showing that there is a lesion of the body of this vertebra which is producing this deformity. Look at that spine move! There is some type of granuloma in that vertebra—either a tuberculosis, a sarcoma, or an osteomyelitis. There is a softening of the body of this vertebra. There must be a granuloma present which presses on the cord and which has caused this inflammatory condition. What it is, we must determine. If it is a granuloma, we do not want to open it; if it is a tuberculosis with a mixed infection, we do not want to open it; we will relieve the pressure, however, so as to free the cord.

We will remove the spinous processes and laminæ of the twelfth dorsal and first and second lumbar vertebræ with bone-cutting and rongeur forceps, being very careful not to injure the cord or its membranes. There is an inflammatory condition in that cord. The left side of the body of the second lumbar vertebra contains a large mass of granulomatous tissue. The cord is pushed over to the right and compressed. Above this point the cord is full and round. Below it is flat. Whether this is sarcoma or tuberculosis I do not know. I think it is a sarcoma. There is a piece of bone perfectly loose. It is just a piece of the process. That piece of bone was compressing the cord. There is a depression in the left side of the cord. It is large enough for me to put my finger into it. The cord above is pulsating. We will remove all this débris with a dull curet, and then free the cord from the inflammatory adhesions. This man has a slight elevation of temperature, but that might be a manifestation of sarcoma. Remember, a rapidly growing sarcoma always causes a rise of temperature. The clinical course of this case has been a very rapid one.

Now the cord is free. We have removed all the laminæ. There is no bone compressing the cord now.

VISITING DOCTOR: What is the prognosis?

DR. MURPHY: Unfavorable. The prognosis depends on the lesion. The prognosis from the operation is unfavorable. So far as the spinal cord is concerned, the prognosis is good. The purpose of this operation is to do a decompression of the spinal cord. That should be done in the early stage, however. He was better able to stand the operation when he came in than he is now. This is the twenty-first. It is now nearly fourteen days since he was able to stand, which is a very rapid course for a spinal cord lesion. I think it is either a granuloma or a sarcoma.

I will ask Dr. Mix to discuss the clinical history of this case from a neurologic standpoint.

DR. MIX: This patient is a Lithuanian who came into the medical service originally running a temperature and looking rather hectic. At first it was thought that he might have pulmonary tuberculosis. He was not in my service; he was in the medical service of a colleague. He was turned over to the surgical service by reason of the fact that very shortly after entering the hospital he began to show inability to move his legs. Being a Lithuanian and unable to speak any English, or we being unable to speak his language, it was very difficult to communicate with him in order to get a history, so most of our diagnosis had to be based on physical signs. From what we learned, the trouble began in January, first with a chill; following the chill there was a period of about ten days or two weeks in which he was better. Subsequent to that he began to have pain. When he arrived at the hospital he was capable of standing on his feet, but was extremely weak in his legs. In the course of a few days time he became totally unable to stand or walk. The paralysis in the lower extremities rapidly developed into a paraplegia.

Physical examination showed paresis of the lower extremities. The anterior crural, tibial, and great sciatic territories were all involved. The quadriceps extensor, sartorius, and rectus femoris were paralyzed on both sides. He had no control of the leg muscles in the anterior and posterior tibial groups. He could not move his toes—flex or extend them— a complete paraplegia.

The next matter of importance was the determination of his sensory disturbance. This was very difficult on account of his inability to speak English. I found, however, that I could learn a few words of his language and succeeded in finding out where he felt and where he did not. We found he had a certain amount of anesthesia over the whole of the lower extremity, corresponding to the distribution of the motor disturbances. We found, also,—which was very interesting,—that there was considerable anesthesia in the supply of the anterior crural nerves; that is to say, the territory of supply of the middle cutaneous and internal saphenous branches of the anterior crural nerve were so involved that the point of a pin could be looped beneath the skin without causing pain. We made this sensory test in order to determine the margin of the anesthesia. We found it to extend up to the point of supply of the ilio-inguinal and iliohypogastric nerves, which nerves come from the first segment of the lumbar cord and make up the first two nerves from the lumbar plexus.

We next turned to the reflexes to see if we could get further information. We found the patellar reflexes absent. We found there were sensory disturbances of the bladder and the rectum and the usual incontinence of the bladder, indicating no sensory lesion in the cord, but an involvement of the vesical filaments to the bladder. We also noted that with the incontinence he had no bed-sores or trophic disturbances such as would occur in a case of cord lesion involving the gray matter.

Since the anesthesia is of the type directing attention to a disturbance of the cord roots rather than to a disturbance of the cord segments, in that it follows definite nerves rather than a segmental distribution of the cord lesions, we are forced to conclude that the lesion is one affecting the cauda rather than the cord. Inasmuch as the upper portions of the lumbar roots are involved, as shown by the involvement of the territory of the anterior crural nerve, we must conclude that the level of the disturbance is as high as the lower end of the conus. This made us localize the process at the first lumbar vertebra. I believe that in this particular case we may relieve him somewhat by operation.

This history is very interesting and typical of all similar cases. The trouble appeared first as a sensory disturbance, followed almost immediately by pain. The pain, as we understand, was more or less severe, and continued for two months, when the motor symptoms began to develop and develop with great rapidity. Such a train of symptoms may come with a tumor of the cord, but does not come with the *ordinary* intradural or extradural tumor. It is more apt to come with a rapidly growing sarcoma or tuberculoma. Within two weeks' time he became almost completely paraplegic.

By reason of the course of the case we were of the opinion that the lesion was an inflammatory granuloma of some type. Next as to the type, we rather leaned to the diagnosis of tuberculosis by reason of the fact that after he had been given an injection of 5 minims of No. 2 tuberculin he obtained a reaction; the diagnosis was made difficult by the fact that at the time he was given the injection he was running a temperature of 101° F., but the very afternoon the injection was given he had a temperature of 103° F., and on the following day it was 102° F. Inasmuch as he had a temperature of 103° F. preceding the test, we are unable to state whether the test showed tuberculosis or not. Examination of his lungs failed to disclose tuberculous focus. Examination of his internal organs failed to disclose tuberculosis there. We, therefore, were compelled merely to suspect the possibility of its being tuberculosis without being able to state definitely that it was. Now, after the operation is done, we are still unable to say what it is, for it might be either a sarcoma or a tuberculoma. The microscopist will have to settle the diagnosis.

Dr. Murphy: This soft débris looks like a necrosis accompanying osteitis, tuberculosis, or a necrosing sarcoma. The height of the temperature makes me believe, however, that it is more likely to be a sarcoma. We have done all we can for this man. We will approximate the cut edges of the muscle aponeurosis with catgut, unite the deeper skin structures with silkworm-gut, and the skin-edges with horsehair. A plain dressing will be applied. No drain; no cast. The patient will be allowed to lie on his back. The spinal column needs no further support than it has now.

[*Microscopic Examination.*—Myeloma with extensive hemorrhage and necrotic area. The patient left the hospital in about six weeks very much emaciated. His paraplegia had not improved. He died about two weeks after leaving the hospital.—Ed.]

APPENDICITIS

A talk by Dr. Norman Bridge, of Los Angeles, Cal., at the Clinic held on Wednesday, June 4, 1913

Dr. Murphy: We have with us today a doctor who was present at my first operation for appendicitis in the Cook County Hospital on March 2, 1889.

This was the first case of appendicitis operated on in America on the then modern basis of opening the peritoneum in the absence of phlegmon or pronounced induration. A paper describing this operation was read by me before the Chicago Medical Society at that time, under the title of "Perityphlitis," in which I reported cases and the results of postmortem observations. The paper was published in "The Western Medical Reporter" for December, 1889. This operation antedated McBurney's operation on the same basis twenty days. The idea of early operation was advanced by Fitz, of Boston, two years preceding that time. Kraft, of Lausanne, had written his able paper and published it in No. 88 of Volkmann's "Beiträge," and it was this paper that gave me the courage to perform my first operation. The conclusions of that paper are practically my present position on appendicitis, with the exception that now all the appendices are removed, except where there is some untoward condition contraindicating that operation. The reason this paper has not been more commonly quoted in the literature is that it was published under the title of "Perityphlitis." Dr. Bridge was in the surgical and medical activities of that time, and made a place in the history of the surgical treatment of this lesion. He was not only in it, but of it, and he helped to bring it rapidly to its present condition. I trust that he will pardon me for calling on him this morning and asking him to say something on appendicitis, because the pathologic conditions have not changed since that time. The treatment has changed with many people, but not with all. I feel he can give you more light than the average man who may have had more experience. I have the pleasure of presenting to you Dr. Norman Bridge, of Los Angeles, California.

DR. BRIDGE: The first case that Dr. Murphy had of appendicitis was not that one in the County Hospital, but one of my own patients who had died. It was a postmortem he made for me, in a case of what we then called perityphlitis. In that case a circumscribed abscess was correctly diagnosed in life, and it was understood perfectly that it was likely to burst into the abdomen and destroy the life, as it did. When that postmortem was made, we both felt a sense of deep chagrin that here was a disease that was destroying annually hundreds of people under the name of perityphlitis, inflammation of the bowels, and general peritonitis, and that nothing had been offered to relieve it or to cure it surgically, and very little medically. Some of the cases, of course, recovered by rest, fomentations, and opiates, which were about all that was done in that day for them, except some harmful things, like feeding and cathartics. But many cases recovered, as they always do and always will, with no treatment.

It seemed to me then that surgeons ought to have sufficient courage and ingenuity to make an operative procedure that would become legitimate for the relief of this condition; and that all internists ought to insist upon it and sustain them. Dr. Murphy was fired with enthusiasm in this behalf, and so, with a few surgical colleagues, notably Drs. Lee and Fenger, soon made an impression upon the professional thought of Chicago.

The surgeons had a hard time during those early years, because the physicians were slow and hazy about their diagnoses in these cases, and when surgeons proposed to open the abdomen for appendicitis, it was thought so unusual and hazardous an operation that usually and properly there was a consultation of doctors. The consultant was likely to be the oldest surgeon in the town, who nearly always opposed so startling a measure as a laparotomy, or opposed it until he could see evidence that the patient was about to die without it. I remember that repeatedly, during those early years, Dr. Murphy and other brave surgeons who were blazing this new trail in science, would ask for a certain internist in consultation rather than a surgeon of a former generation, and because they knew the internist was committed to the doctrine that in these cases the patient could probably be relieved and saved if there was courage enough in the surgeon and common sense enough in the patient and his family.

A few internists early discovered that not only was appendicitis a surgical affection, but that they might have to urge the surgeons to operate. At a congress of physicians and surgeons in Washington not long afterward, when this subject was under discussion by both the physicians and the surgeons, the strongest ground in favor of operations was taken by the physicians. Some of the surgeons showed symptoms of fear for their percentages of deaths, and trembled at the thought of being criticized for doing an extra-hazardous operation that might be unsuccessful.

In that decad, more than now, the operations were delayed until after the abscess had formed. Then sometimes, when the surgeon opened an abscess, he felt bound to hunt for the appendix; but it was soon found that that was both hazardous and unnecessary, and that the abscess should be drained only at the first operation. Then, little by little, the surgeons got the opportunity, and the courage of their experience, to open the abdomen and make the operation early, and the early operation has been justified by results to the highest degree. Now we have the rule, which is widely accepted, that immediately after making a diagnosis of appendicitis the operation should be done if the surgeon is competent. But this rule is not as universally accepted as it ought to be. There are many reasons for this: One is the fact, known of all doctors and many lay people, that a large proportion of cases, with rest and good nursing, recover or appear to recover; another is the dread of a surgical operation in one's family even when it is a physician's family; another is the belief, somewhat prevalent among lay people, that surgeons do many appendix operations that are unnecessary. So many cases are left to recover or to die without surgical aid, or with it too late.

Of course, any number of people may recover from this disease without operation, but no human being can tell which patient is going to have a perforation and then an abscess or general peritonitis. Nor is there now any ground for the hope that means will be discovered whereby we can know without opening the abdomen the cases that, left to themselves, will, and those that will not, pass on to perforation and pus. The reasons for this are the differing physiologic equations of the patient, their varying conditions of health, their differing powers of resistance to various microörganisms, the differing physical and chemical causes in the appendix itself, and the anatomic variations of the organs in different patients. These uncertainties will make it forever impossible to settle, without operation, this most vital question. I know that men have believed and argued that they are able to make the distinction, and learned medical societies have wrangled over this question almost interminably, but there is only one safe rule—that is, to operate promptly on a diagnosis being made. With a modern surgeon and the surgical facilities of a modern hospital, there should be no difficulty, and by this course the danger to life will be minimized.

Dr. Murphy has done much toward perfecting the technic of the operation; other surgeons have done much, so that now, in early operations, the mortality is substantially nil. By comparison with the present mortality from such operations, the death-rate in the earlier years was frightful.

We internists have almost as large a responsibility as the surgeons themselves. We ought not only to seek to have every appendicitis case operated early, but we ought, for numerous other conditions and mysteries in the abdomen, to insist on exploratory laparotomy when it can be done by a surgeon of large experience. I am not unmindful of the embarrassment with which one discovers, after such an operation, that it was not necessary, except for the purpose of exclusion, and that the patient and his friends may blame both the physician and the surgeon, but I would rather half a dozen patients have exploratory, harmless laparotomy with nothing to remedy, than allow one perforative appendicitis to go on to death without an operation that might save his life.

The diagnosis is a matter of much perplexity, especially for many of the internists. Some of us need to revise our notions of the symptomatology. We have the habit of looking for certain signs and symptoms supposed to be typical, like sudden pain in the lower right quadrant, tenderness, tension, nausea, fever, and leukocytosis. But appendicitis is as atypical as typhoid fever; the symptoms vary, often some are absent, and they may be mixed in the most erratic manner—pain may be almost absent in the appendix and amount to agony in the epigastrium; the appendix may be in the wrong place; there may be little tenderness in the appendix itself; there may be only a trifle of fever, and no abdominal muscular tension whatever. But enough of these signs and symptoms are usually present to make a diagnosis if we do not set our minds against believing the disease possible. With these symptoms present, the presumption of appendicitis is always warranted, and positive evidence is required to exclude it.

After a recovery from an attack under medical or expectant treatment, beware of lingering symptoms of discomfort or tenderness, or sensitiveness to physical jars, or even unusual constipation. Such conditions often handicap the patient for many months, and they demand surgical relief.

ON EARLY OPERATION IN PERITYPHLITIS

Abstract of a paper published by Dr. Murphy, February 26, 1890. Reprinted to show striking similarity between Dr. Murphy's teaching of today and that of twenty-five years ago.

Within the last few years the forms of perityphlitis, in which there was an absence of phlegmon of the abdominal wall, entirely came within the domain of internal medicine. We will endeavor to demonstrate that *perityphlitis vermicularis belongs always to the province of surgery*, and that its dangers are avoidable and its recurrence preventable only by surgical interference. This may appear a radical statement, but it is sustained by two facts which we may consider as positively proved: (1) That perityphlitis is *always* accompanied by the development of a pus cavity. (2) That with the present plan of treatment the patient is more liable to a recurrence of the disease.

That perityphlitis is a more serious disease than is generally supposed can be demonstrated by examining the postmortem reports of the great hospitals of the world. In our limited experience we have made autopsies on at least three cases within the past three years, which showed that fatal peritonitis was caused by the rupture of the perityphlitic abscess into the peritoneal cavity, where its existence had not been previously diagnosticated.

In making our prognosis we must not content ourselves by simply saying such a per cent. have successfully recovered from the first attack. We must further consider how many patients succumb who have recurrent attacks of the disease within five, ten, or fifteen years. The dangers are:

(*a*) Perforation of the appendix, permitting escape of its contents into the peritoneal cavity directly or indirectly by a rupture of the circumscribed suppuration.

(*b*) Where the abscess is extraperitoneal by the formation of a septic phlegmonous cellulitis and death from sepsis.

(*c*) By the emptying of the contents of the abscess into some of the neighboring organs or cavities, as the bladder, kidney, or thoracic cavity.

The highest rate of mortality is reached in the first, notwithstanding the fact that inflammatory adhesions are rapidly formed for the protection of the peritoneal cavity. Were it not that in many cases the adhesions are accidentally ruptured either by a too violent movement of the body (Meyers, Langdown, Carson), incautious manipulation, injudicious diet, persistent vomiting, or too energetic catharsis, the rate of mortality would be very much diminished, as the pus would escape into the cecum by the ulceration from without inward. Further, the peritoneal cavity is protected by the anatomic arrangement that the appendix is furnished with a small mesentery which holds it in position and very materially limits its mobility. In the absence of this condition the danger of peritonitis is much increased.

Perityphlitis treated on the expectant plan is a frequently recurring disease.

One of our cases, a girl of seventeen, had three attacks within a year; in another, that of a young man of twenty years, four attacks were experienced in a period of eighteen months. A well-known case is that of one of the leading physicians in this city who was incapacitated for a period of two years by repeated attacks of this disease. Every recurrence can prove fatal. Thus attacks of perityphlitis are not so harmless as has been generally supposed.

The preponderance of evidence, as furnished by recent observers, tends to prove that in all cases of perityphlitis vermicularis the disease is attended by the development of a pus-cavity. Kraft, in his 106 cases of this affection, in which the diagnosis was verified either through autopsy or direct incision into the abscess cavity, found that in all of them greater or smaller accumulations of pus were present at the seat of the appendix or cecum. Of the 106 cases, 84 had autopsies, and 84 times an abscess was found in the ileocecal region. Eight times he operated *in the absence of fluctuation, redness, or swelling of the abdominal wall, and 8 times* did he find pus. In the remaining 14 cases the abscess emptied into the cecum or other organs.

Of the 13 cases which have come under our observation, in which the diagnosis was verified either through the autopsy or by incision, pus was found in every case. In the face of such evidence as this, proving the presence of pus, it behooves us to consider what protection is afforded the patient against the danger of an attack or its recurrence by the so-called expectant plan of treatment. We order rest, opium, liquid diet, etc., hoping by this means to favor the formation of adhesions, and so protect the peritoneal cavity and favor the exit of the pus by way of the intestines or some other organ, or through an opening in the abdominal wall. How much is gained by this can be estimated by the statistics furnished by With, who reports 30 cases treated with purgatives and injections, in which the death-rate was 40 per cent.; subsequently 50 cases treated with opium, rest, and a suitable diet, in which the death-rate was 16 per cent., with an average death-rate of 28 per cent. under medical treatment.

This leads us to consider: *Are we doing our duty to our patients by allowing them to take such chances?* We think not. Why should pus in *this* locality be allowed unaided to find its favorable or unfavorable exit, in contradistinction to the well-established rule to promptly aid its escape in all *other* parts of the body where accessible? Who here has not seen a similar case to this? The patient is taken suddenly ill; complains of pains in the abdomen, has vomiting, a rapid, feeble pulse, and a pinched, anxious expression of countenance. Examination reveals the abdomen to be uniformly distended and sensitive—in short, with all the symptoms of acute peritonitis, usually terminating fatally on the third or fourth day. *Were we permitted to make autopsies on all of the cases presenting the above history, we would find that a large percentage of them were produced by the rupture of perityphlitic abscess into the peritoneal cavity.*

Mikulicz, in 1887, said that perforation of the vermiform appendix with purulent peritonitis is of common occurrence. Within the last seven months we have had two autopsies on similar cases at the Cook County Hospital. The fact that we are not allowed to make postmortems in all cases of peritonitis, makes it difficult to draw any definite conclusions as to the relative frequency of this as a causative agent, while the cases in which spontaneous opening occurs through the abdominal wall, are generally reported on account of their successful issue. The unsuccessful ones lie buried in oblivion with the victims. Where we meet with a case in which the symptoms of general peritonitis are present, we should examine more closely into the history of the patient, past and present, and we will frequently find that the seizure is accompanied by pain and swelling in the right iliac region, which has existed for days, months, or even years, previous to the present attack, and that a large percentage of the cases of general peritonitis is the direct sequence of perityphlitis.

Failure to make a correct diagnosis is usually dependent upon assuming the case to be a primary general peritonitis, without ascertaining minutely the previous history and making a thorough physical examination and considering the relative local intensity of the inflammatory symptoms and physical signs. The induration so commonly found in the right iliac region is frequently overlooked on account of the tympanites diffused over the whole abdominal region. It may also be obscured through the generation of gas in the abscess cavity lying beneath. With careful percussion, dulness may be detected over the region primarily involved. Should the abscess happily open into the cecum, so will the result of the case be favorable. This does not occur through the tract of the vermiform appendix, as is commonly supposed, but ulcerates from without inward into the lumen of the cecum (Kraft). Should it unfortunately open into the thorax, there ensues an empyema; into the portal vein, a pyophlebitis; into the bladder, a septic cystitis. Perityphlitis can, therefore, end in three ways: (1) As a general peritonitis. (2) It may burst into the neighboring organs and produce as dangerous a complication as the original disease. (3) It may open through the abdominal wall. The hope that perityphlitic abscesses can disappear by absorption is a delusive one.

That the cause of perityphlitic abscesses is perforation of the appendix is sustained by the investigations of Matterstock, who found in 146 autopsies on adults of cases of perityphlitis that there were 132 perforations of the appendix. In 49 autopsies on children for the same affection perforation was present in 27 cases. Fenwick, in 129 autopsies, found 113 perforations of the appendix, and in 16 cases an abscess in its neighborhood. Weir found, on investigating 100 autopsies of this disease taken from the Index Medicus, the vermiform appendix was reported as having been perforated 84 times. L. Gautier found in 50 autopsies only 2 cases in which perforation was absent, making in all 465 autopsies with 324 perforations (about 70 per cent.).

In all cases where there was not an exact examination of the pathologic condition we can reasonably assume that there existed a perforation of the appendix. It is recorded in cases of perityphlitis that the cecum is frequently perforated from within outward. But Kraft is convinced that if a careful examination of the opening be made, it would be seen that the *perforation occurs from without inward*, and not *from within outward*, showing that the opening in the cecum was not caused by an ulceration of the mucous membrane, but by an ulceration of the abscess wall into the cecum. Often is the vermiform appendix not only perforated, but part of it entirely destroyed, as in two of our cases. Perforation usually occurs at the free end, but when it occurs in the middle, for example, the ulceration is circular and the appendix can be amputated as in one of our cases.

The cause of the perforation is found to be most commonly fecal stones. When we consider the difficulty of finding in such a quantity of pus and effusion a body as small and perhaps smaller than a bean, we can see how readily it may be overlooked. It is also a fact that it may be broken into small particles. The stone may be found within the pus-cavity in the opening of the vermiform appendix or entirely within its lumen. It is a small, bean-shaped body, rounded off at both ends, usually smooth on the surface, and varies in sizes from that of a small pea to that of a peanut; some of them are hard, others soft and friable. The center is usually of a yellowish gray color.

The frequency of fecal stones is contrary to the generally accepted idea that perityphlitis is produced by the accidental presence of cherry-stones and seeds in the vermiform appendix. Different pathologic and anatomic elements combine to produce retention of the offending material within the appendix, which hangs upon the inner and under portion of the cecum; it extends from its attachment to the cecum directly upward and a little inward. It presents great difference in form and position. As a rule, it has its own mesentery which extends from one-half to one-third its length. Often this is absent, and thereby the formation of a fecal stone is more liable to occur at the point of union of the lumen of the vermiform appendix with that of the cecum. It varies in length from one to three and one-half inches. We present a specimen 3¼ inches. The vermiform appendix is very often adherent to the surrounding tissues. The abnormalities in the development of Gerlach's valve is one of the potent elements in producing retention of feces (Eichhorst).

The absorbing surface of the appendix is great in comparison with its lumen, for which reason the feces within became hard, and from the secretion of the mucous membrane the deposit of lime is accelerated and layer by layer deposits itself upon the nucleus until its size is sufficiently large to occlude the lumen. This produces pressure atrophy and perforation. Thus perforation will take place—(1) Into the peritoneal cavity if no adhesions are formed; (2) into the cecum if the vermiform appendix is adherent to the cecum; (3) into the pelvic cellular tissue if the vermiform appendix is adherent to the parietal peritoneum; and (4) but very rarely through the original cavity of the vermiform appendix into the cecum. Let us now consider the conditions of the ileocecal region in autopsies upon this class of cases. The omentum in such cases is thickened in this region and is adherent to the intestines which it covers. If the omentum is elevated and the intestines separated from each other, it will be observed that there are several abscesses divided from each other by fibrous layers; then there will be an abscess in the lesser pelvis separated from the retrocecal abscess by adhesions. The abscesses are usually not voluminous and contain a fecal pus which usually has an intensely offensive odor. Kraft says that all investigators are united in stating that a portion of the appendix is not covered by peritoneum, but floats in the retrocecal cellular tissue, although it cannot be contradicted that there are frequent exceptions to this rule and that the appendix often lies entirely in the peritoneal cavity. This is contrary to the observations of Bardeleben (1849), Luschka (1861), and latterly Weir, Richardson, Treves, and Hektoen.

All the latter agree that the vermiform appendix in the great majority of cases is within the peritoneal cavity. This discrepancy may be accounted for by the different interpretation of these observers as to the manner in which the peritoneum is reflected around the vermiform appendix at its base, while it appears to be within and completely encircled by peritoneum at its mesenteric attachment, about one-eighth of its entire circumference is not covered by that membrane. *As the pus within the base of the appendix accumulates it meets the least resistance at this uncovered point, and therefore ruptures through this locus minoris resistentiæ into the pelvic cellular tissue, making the perityphlitic abscess extraperitoneal.*

It is of great importance in considering the pathology of perityphlitis whether the appendix be intra- or extraperitoneal. It is of still greater importance to know whether the abscess is formed within or without the peritoneal cavity. Adhesions can and frequently do take place between the vermiform appendix and the parietal peritoneum, so that ulceration continues from the wall of the appendix on through the parietal peritoneum into the cellular tissue, producing an extraperitoneal abscess, as was the condition in numbers of our cases. Weir states that the abscess is always intraperitoneal, but he draws his conclusions from the result of 100 autopsies. This is misleading, as the extraperitoneal abscesses are rarely fatal, and the fatal cases of perityphlitis almost invariably belong to the intraperitoneal variety. As a result of observation in our operations we must demur from his conclusions on this subject. If it perforates extraperitoneal, the cellular tissue becomes infiltrated, suppurates, and is surrounded by adhesions. *In operating on this class of cases, as soon as we reached the subperitoneal cellular tissue, and often before, we found evidences of plastic inflammation. On separating the exudate we found within it a small pus-cavity. If the perforation of the appendix is intraperitoneal, that is, the abscess is formed within the peritoneal cavity, we will have to incise the peritoneum before we come upon the evidences of inflammation.*

We do not consider in this paper the cases in which fluctuation is present, for those have always been successfully operated upon. *We wish to call the attention of the profession to cases where there is only a small induration in the ileocecal region with the accompanying symptoms of perityphlitis before there is the slightest evidence of fluctuation or phlegmon in the abdominal wall.* It is difficult to give the exact indications for the proper time to operate when the symptoms are slight, for both the patient and the surgeon are reluctant to resort to operative interference. To the future we will leave the answer to this question. As yet the number of cases operated on have been too small to lay down any definite rule, notwithstanding it is our conviction that before many years every case of perityphlitis when diagnosticated will be immediately opened, the appendix ligated, if possible, and amputated. This operation gives the only guarantee that a patient can have of safety from the impending danger of the disease and security against its return. It must be remembered that in making the examination the greatest caution should be exercised in palpation and percussion, as we know from our experience in making autopsies that slight pressure can rupture the adhesions and the whole contents of the pus-cavity escape into the peritoneum.

Cathartics and clysters should also be studiously avoided, as they are liable to have disastrous effects.

The most positive symptoms indicating the existence of this disease are the following: Pain is always present, begins suddenly, is of a lancinating, pulsating character. The first day or two it may be indefinite, being felt all over the abdominal region, but soon it locates itself in the right iliac fossa. Occasionally the pain radiates along the course of the anterior crural nerve, the thigh being held in a flexed position. The attack is usually ushered in with a chill. The patients are ordinarily young, strong persons, who frequently state that they have had previous similar attacks. Symptoms of intestinal obstruction are commonly present. Temperature ranges from 102° F. to 104.5° F., continuing without interruption for the first four or five days; then it suddenly falls to 101° F. or even to 99.5° F., and continues so for days unless there be an invasion of new tissue, when we have again the symptoms of infection, high temperature, etc. The fact that the temperature remains from 99.5° F. to 100.5° F. and the absence of chills and sweats, are no indication that suppuration is not present. On the contrary, in all our cases the absence of chills and sweats was most marked, and yet in every case there was a pus-cavity found. *The one positive sign was the presence of a small induration in the right ileocecal region, and in this induration we invariably found pus.* The reason for the fall of temperature and the absence of other symptoms of sepsis, is the protection afforded the general circulation by the plastic infiltrations of the wall of the abscess. The absence of dulness is not to be relied upon, as occasionally gas is generated within the abscess cavity. An induration of the pelvic cellular tissue can be found in a small number of cases by examination through the rectum.

In making the diagnosis we feel constrained to insist that the use of the hypodermic needle in these cases should be abandoned. Bull, in his paper, while he suggests its use, admits that it is not without danger. If the abscess is within the peritoneal cavity and we introduce a hypodermic needle, there is danger of rupturing its walls and the escape of its contents, or the escape of some particles on the withdrawal of the needle into the peritoneal cavity enhances the danger of setting up a fatal peritonitis. Why increase the risk of this accident by the use of the needle when you are free to make your incision as deep as may be necessary and yet without increasing the danger materially? The use of the hypodermic needle in this disease is, like many surgical procedures, ostrich-like, since we hope to avoid danger and are sure not to see it when it occurs.

We must not depend upon the number of days from the beginning of the attack as a guide to operative interference. The cases differ so widely that often after forty-eight hours from the onset it is already too late; in other cases weeks or months may elapse before an operation is demanded. Of the 76 fatal cases collected by Fitz, 56 per cent. died from perforation in the first week, 31 in the second, 4 in the third and fourth. His statistics show how early a fatal termination *can* take place and how *imperative* an early operation is if we would lessen the risk of losing our patients.

In general, it can be said that as soon as the diagnosis is made too early an operation is impossible; it frequently occurs even then that it is too late.

The Technic of the Operation.—As soon as it is considered settled that the diagnosis is correct, give a hypodermic injection of morphin with atropin, anesthetize the patient, make the typical incision for ligation of the right common iliac, taking precaution to have the incision sufficiently long. When the subperitoneal cellular tissues are exposed, if the case be of the intraperitoneal variety, there will be no evidences of inflammation until after we have opened the peritoneum; not the slightest sign of inflammation will exist extraperitoneal. It is a safe guide to dissect down over the center of the induration until after you have passed the peritoneum; then in the intraperitoneal variety separate the adherent intestines, and you will come into the abscess cavity. This should be cleansed, the fecal stone or other foreign body removed. If the vermiform appendix is floating in the abscess cavity, it should be ligated at its base and excised, a drainage-tube inserted, and a tampon of gauze around the drainage-tube from the bottom to the surface to protect the peritoneal cavity from the pus. In the extraperitoneal variety the abscess cavity should be washed out, the foreign body with the vermiform appendix removed, unless the latter is adherent. Great precaution should be taken in exploring the cavity, because the slightest laceration of the adhesions may admit the septic contents into the peritoneal cavity and cause a fatal peritonitis. If the peritoneum should be opened accidentally in the extraperitoneal variety, it should be united with sutures and the abscess treated as before. The tampon should be allowed to remain for two or three days, as frequent disturbance of it increases the danger of infection and laceration of adhesions. If the peritoneum is not opened, the operation is *per se* almost without danger, and is a certain preventive against the return of the disease, providing the appendix is amputated.

LAMINECTOMY FOR RECURRENT ENDOTHELIOMA OF SPINAL CORD—THIRD OPERATION

HISTORY

April 25, 1913.

Dr. J. B. Murphy,
104 S. Michigan Avenue, Chicago.

My Dear Dr. Murphy: Yesterday I examined Mrs. W. L. P., now at Mercy Hospital. During the last three or four months symptoms of spinal-cord pressure have recurred, and she is now very much in the same condition that she presented previous to the second operation, a year ago. This second operation was not followed by the same amount of recovery that succeeded the first. Still, aside from a feeling of clumsiness and numbness in her feet, she was fairly well and able to look after her household cares.

The stiffness and numbness in the lower extremities, which began to increase three or four months ago, have progressed steadily, until now she walks with very great difficulty and in a very rigid manner. She also uses her hands a little less dextrously than before.

I find that sensation is disturbed from the third rib downward in front, and from about the third dorsal vertebra downward behind. This area extends on to the inner borders of arms, forearms, and hands, embracing the little finger and a corresponding strip upward on the forearm, etc., as shown by inclosed diagram. The line of sensory change is a little difficult to determine, and varies slightly when approached from below as compared with the point indicated when approached from above, *i. e.*, it is not absolute.

It indicates that the spinal symptoms are due to a lesion somewhat lower than at the time of the first operation. As I am not familiar with her symptoms at the time of the second operation, I cannot say whether it corresponds to those or shows a downward involvement, but at any rate it will be necessary to explore at least as low as the bodies of the first and second dorsal vertebræ. As I presume this to be below the level of the second operation, it may be inferred that we are dealing with a new-growth or a recurring growth, rather than with scar tissue, etc.

I find the reflexes all very much exaggerated excepting at the elbow. In the lower extremities exaggerated knee-jerk, very slight ankle-clonus, and Babinski are present on both sides. In the upper extremities wrist-jerks are exaggerated, elbow-jerks diminished.

I should be very glad to know what is done and with what result.

Very truly yours,
(Signed) A. CHURCH.

COMMENTS AND OPERATION

DR. MURPHY: You will have to follow this case through the two previous histories. The first history was written in 1910. The second history was written in 1912. We did the first operation August 29, 1910, and the second operation May 17, 1912.

Patient steadily improved for seven or eight months after leaving hospital following operation on May 17, 1912. She could walk 17 or 18 blocks at a time. About four months ago she began to have numbness in the fourth and fifth fingers of the right hand. Shortly afterward the right foot began to get numb; then the left foot. She thinks the symptoms are not as severe as they were when she was operated on one year ago. Dr. Church thinks the lesion is located at the third dorsal vertebra.

[NOTE.—The previous history of this case and the operation were fully recorded in the CLINICS of October, 1912, page 695.—ED.]

DR. MURPHY: In the last examination made by Dr. Church he located the lesion as extending as low down as the third dorsal. You can imagine how very unpleasant it is to have to operate where the landmarks are destroyed and there is nothing but cicatricial tissue.

We will make a rather long incision, extending from the spinous process of the third cervical to that of the fourth dorsal vertebra. The bleeding here is a good deal like the bleeding in the dura. If you just wait and compress the tissues, the bleeding finally subsides. It is a venous bleeding. We can get a splendid exposure by retracting the tissues with sharp hook-retractors. In the previous operations we removed the spinous processes and laminæ of the third to the eighth cervical.

Tumors Involving the Spinal Cord

1. Dermoids—rare (White's case).
2. Sarcoma:
    a. Spindle-cell.
    b. Round-cell.
    c. Myxosarcoma.
    d. Osseous, invading.
3. Carcinoma:
    a. Intradural.
    b. Extradural.
4. Myeloma—body of vertebræ.
5. Glioma (Ressinger, 19 cases).
    (More rare than in brain, 1: 20.)
6. Neuroma.
7. Psammomata of the pia.
    Endothelioma of dura or pia.
8. Cysts:
    Echinococcus { Intradural. / Extradural.
        Body of vertebræ.
        Connective tissue and invading.
        Circumscribed of the meninges.

You will note in the tabulation of the spinal cord tumors given on the board that we have endeavored to outline the various types of neoplasms that occur in the cord and near the cord. We have had rather an extensive experience with these tumors, and, so far as the non-malignant variety is concerned, we are becoming quite hopeful. As to the malignant variety of tumors, we are becoming less hopeful. We are getting more results from the decompression operations than from the radical treatment, because the decompression is applicable to the non-malignant variety and is often curative.

I have removed the spinous processes of the first three dorsal vertebræ with the rectangular bone-cutting forceps from below upward, and we have a very nice exposure of the cord.

Now I am coming to a mass here where the dura is adherent to the scar tissue resulting from the previous operations. I have secured a good exposure without disturbing the cord. Now we will open the dura. I can feel a resistance here. Whether that is the resistance of the fixation of the dura or the resistance of the neoplasm I do not know.

There is no irregularity or elevation to be felt anywhere. There is something to be felt within the dura, on a level with the fourth cervical vertebra, firm and compressing the cord. There is nothing to be seen or felt outside the dura. I have a splendid exposure of the cord. I am well up above the fifth cervical. There is the neoplasm. It looks like a little egg. That shows how erroneous these symptoms were as to the location of the tumor. It is on the right side of the median line. It is about as large as a bluebird's egg apparently, and seems to be encapsulated. I am going to enucleate it. It attacked the spinal nerve-roots originally, just about the level of the fourth cervical. I am proceeding very cautiously because I am near to vital tissue, and a little trauma can do much harm.

I think now I have removed the capsule with the tumor, but I am going to cauterize its bed with formalin and glycerin and then pack the wound with iodoform to stimulate connective-tissue formation. What I will do today is to protect the cord from pressure in the future. I will suture the muscle in between the edges of the divided dura, so as to give this point a permanent decompression, that is, to permit of the backward expansion of the cord if a new tumor should form. This I believe is an original procedure. If the tumor grows again, it will have to grow in the direction of least resistance and come out backward, thus preventing a crushing of the cord. I am suturing a muscle-flap in there for the entire length so that the edges of the dura cannot again become united. Many surgeons are using muscle as a substitute for the dura in brain operations, in decompressions necessitating removal of the dura.

I am going to give this patient some *x*-ray treatments this time. These endotheliomata are usually not malignant as far as metastasis is concerned. They recur locally sometimes and compress the cord, therefore it is vital for us to protect this cord from pressure, that is, to give the tumor a chance to get out when it grows the next time. It was between the columns of Goll and Burdach. We were able to separate these two columns in the original operation and get the tumor out without leaving a permanent defect of sensation that was recognizable by the patient or on examination by the doctor.

Let the record show that the tumor was located in the same position between these two columns as the primary tumor, but higher up; that it was enucleated within its capsule; that there was but little bleeding; that the tumor was situated to the right of the midline, with one-fourth of the cord displaced to the right and three-fourths to the left. The operation was done with much greater ease than the two previous operations.

After once getting to the cord and allowing the cicatricial tissue to fall back and elevating the dura, it was not difficult to locate the tumor. We just split the tissues and brought it out.

We close the wound with through-and-through silkworm-gut sutures. We did not have to ligate any bleeding points because compression controlled what little hemorrhage there was.

Let the record show that one Victor Horsley sponge was inserted and reported returned.

No cast will be put on, just a plain dressing, and she will be allowed to lie flat on her back. She should have no trouble if that field of operation remains sterile, which we believe it will. Remember we were in the true cord, but we believe that not enough of the axons were cut or destroyed by compression to render a comparatively complete regeneration impossible or improbable.

[Microscopic examination showed the tumor to be an endothelioma. The patient left the hospital early in the fifth week, free from pain. Sensation and motion returned rapidly, and she is again as well as she was immediately after the first and second operations. *Operations for tumors of the cord should be performed before paraplegia occurs*, as the axons that are destroyed are permanently out of commission, while in the partial paraplegias the axons are only compressed, not degenerated; transmission is suspended, and can return completely. There is no excuse for allowing a non-malignant cord tumor to develop to complete paraplegia, except on the basis of incompetence and inefficiency on the part of the physician or surgeon in charge.]

GLIOMA OF RIGHT CEREBELLAR LOBE—PATIENT KEPT BREATHING WITH PULMOTOR FOR THIRTY-FOUR HOURS

HISTORY

Patient, female, aged twelve, was operated on by us and a cerebellar decompression done six years ago from which she recovered entirely. Two years ago, following an attack of tonsillitis, she had attacks of severe pain in the occipital region and vomiting. She recovered and was well until six weeks ago (about April 1, 1913), when she began to complain of earache. The ear never discharged. She had attacks of earache every third day at first. These attacks became more frequent, occurring every other day, then every day for five weeks. Two weeks ago she began having attacks of severe pain in the occipital region. These attacks occurred every other day at first, but are almost continuous for the past day or two. Saturday, April 12th, she had an attack of vomiting. Sunday, April 13th, she felt well. On Monday she had another attack of pain with vomiting at noon. Tuesday, April 15th, she felt well. Wednesday the pain was again severe and she vomited twice. Thursday she had severe pain and vomited several times. She has a choked disc in both eyes.

COMMENTS AND OPERATION

Dr. Murphy (May 12, 1913): This is a case of tumor at the center of respiration in the brain, in which the patient was being kept alive by the pulmotor. I operated on her six years ago for a cerebellar tumor, doing an occipital decompression, and she got perfectly well. She is now nearly thirteen years of age. The first time we operated she had all the symptoms of a cerebral lesion. Since then she developed into a splendid girl. She was perfectly normal in every way. About six weeks ago she got an infection in her throat,—a tonsillitis,—had some temperature elevation, began to get headaches again, became nauseated, and developed a choked disc. She was brought to the hospital from El Paso, Texas, a week ago. Her symptoms all subsided until last Thursday, when the headaches became more severe and she became more nauseated. On Friday she improved and had a fairly good day. On Saturday she had the best day that she had since she had tonsillitis. She had a leukocyte count of 11,000. She got along all right until Saturday evening, about 8 o'clock, when she had a convulsion and became unconscious. It was the first convulsion in this attack. She regained consciousness. She had convulsions before we did the decompression six and one-half years ago. She went along apparently well until 7.30 Sunday morning, when she began to show difficulty in breathing. She became cyanotic, and finally all respiratory efforts ceased and she appeared to be dead, but her heart was still beating. We started artificial respiration in a regular, systematic way. Her pupils contracted somewhat, but she did not show any efforts at breathing. We kept up the artificial respiration for two and one-half hours, until we got a pulmotor. We kept her alive with the pulmotor for thirty-four hours. Her heart beat right straight along, perfectly regular. Her color was splendid. Suddenly she became blanched and died Monday evening about 6 o'clock. Her heart-beat was 84 yesterday morning, thirty hours after the stoppage of respiration. She had no reflexes from the rectum, no reflexes from the bladder, and no reflexes from the pupils. She was dead except for the heart-beat. The pulmotor kept the lungs going.

The pulmotor is a wonderful machine. It is almost human in its working. If the air is not going into the lung, it stops working.

We operated on her again Sunday evening to relieve the pressure on the brain. The intern will read his notes on the operation. Operation Sunday, May 11, 1913: Incision over trachea. Isthmus of thyroid elevated; low tracheotomy done and rubber tube put in trachea and attached to pulmotor. No anesthesia. Incision over cerebellum and periosteum around old scar elevated and decompression done. No pulsation of dura. Enormous pres-

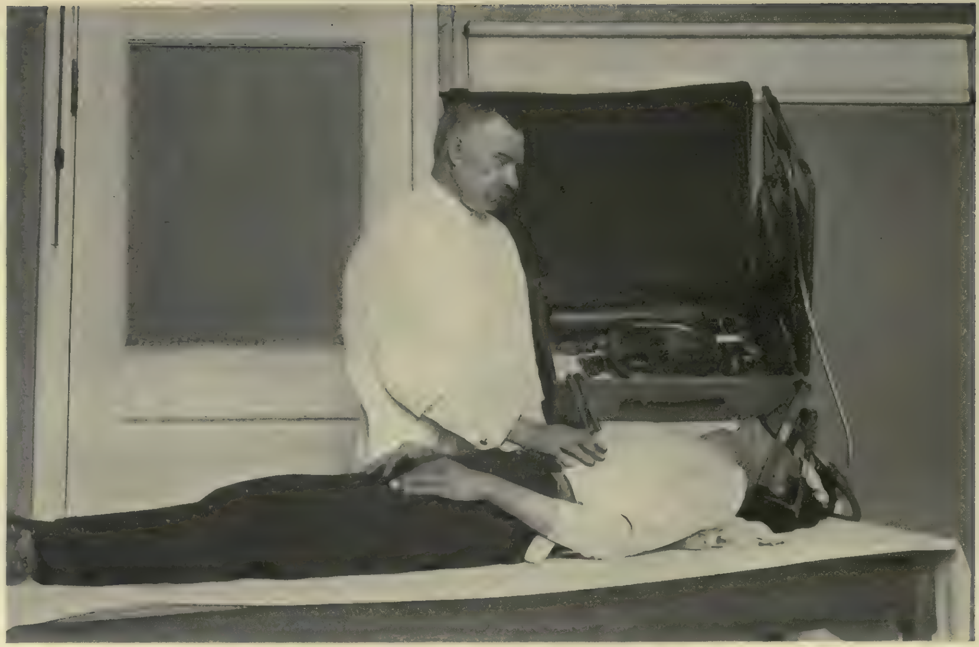

Fig. 176.—Showing the pulmotor in action, with the patient recumbent, and the apparatus set up on his right.

sure over cerebellum. Dura opened and pressure relieved. Wound closed.

We performed an autopsy on her last night and found that she had a small tumor in the ependyma, not larger than a walnut, just at the opening of the fourth ventricle, at the base of the respiratory center, causing a paralysis of respiration. It is the second case of this kind I have seen. The first case I saw when I was an intern. The man had been in Union Park, got on a Madison Street car, rode a couple of blocks, when he fell over. He was brought into Cook County Hospital. He looked as though he had taken poison with suicidal intent. We kept up artificial respiration for twelve hours, until the heart stopped beating. We held an autopsy and he had a tumor at the same place as this patient. You cannot operate on these cases.

The pulmotor is too expensive a machine for common use, but we are working out a plan to run it for 3 per cent. of what it costs to run it now. By referring to the article on the Surgery of the Lung that I wrote in 1898, you will find that I showed that more than 3 per cent. of oxygen in the air is an absolute waste; that the lungs will take up as much from 3 per cent. additional oxygen in the air as they will from 100 per cent.; therefore you can use 3 per cent. of additional oxygen and 97 per cent. of compressed air and get apparently the same results.

I will ask Dr. Mix to tell you what was found at the postmortem.

Postmortem Examination.—By Dr. Charles Louis Mix and Dr. Walter McGuire, May 12, 1913.

The most notable thing in removing the calvarium was the thinness of the bone. Its under surface was absorbed to conform with the convolutions. In many places the bone was transparent. As soon as the calvarium was removed the brain was found to be remarkably soft and edematous, making its removal extremely difficult. The brain was immediately put in a 5 per cent. formalin solution, and subsequently examined by Professor F. R. Zeit, whose report was as follows: The dura was markedly thickened, the convolutions being just visible through it. The dura is only slightly adherent to the brain. The longitudinal sinus contains some dark-colored blood. The convolutions of the brain were very markedly flattened, suggesting an edema. The blood-vessels of the pia mater were injected with dark-colored blood. The corpus callosum is extremely soft and easily torn. The ependyma of the lateral ventricle is smooth, mirror-like, and glistening. No ependymitis is present. Both lateral ventricles are slightly dilated. The consistence of the cerebral hemisphere is much diminished because of the edema. The consistence of the cerebellar hemisphere is also much decreased. The floor of the fourth ventricle contains a mucoid material. The aqueduct of Sylvius is not especially dilated. The ependyma of the fourth ventricle is markedly injected.

In the right cerebellar hemisphere, at the base, is a tumor the size of an olive. The tumor is rounded and firm in consistence in its anterior extremity, and posteriorly it shows an area of degeneration. The degenerated area appears somewhat like caseous degeneration, and extends to the ependyma, from which the tumor seems to originate. The base of the tumor encroaches on the roof of the fourth ventricle. The length of the tumor is 4.5 cm. and it is 1.8 cm. in diameter. The anterior portion of the tumor is olive-shaped. The posterior portion is degenerated and merges into the adjacent cerebellar tissue and ependyma. It is possible to loosen the anterior portion of the tumor mass from the cerebrum and to shell it out. From its gross appearance it suggests a sarcoma with a degenerated area extending into the floor of the fourth ventricle. The fourth ventricle is very much dilated, being about twice its normal size.

On making serial sections of the cerebral hemisphere no edematous areas, softening, or hemorrhages were found in the caudate nucleus, the lenticular nucleus, or optic thalamus. Both right and left internal capsules were normal. No tumor, hemorrhage, or softened area is to be found in the white matter of either the right or the left cerebral hemisphere.

Turning to the cerebellum, a slight leptomeningitis is found in the posterior fossa, apparently recent in type. On section of the right cerebellum a cyst is found posterior to the tumor. The diameter of the cyst is 5 cm., and it contains a mucoid material

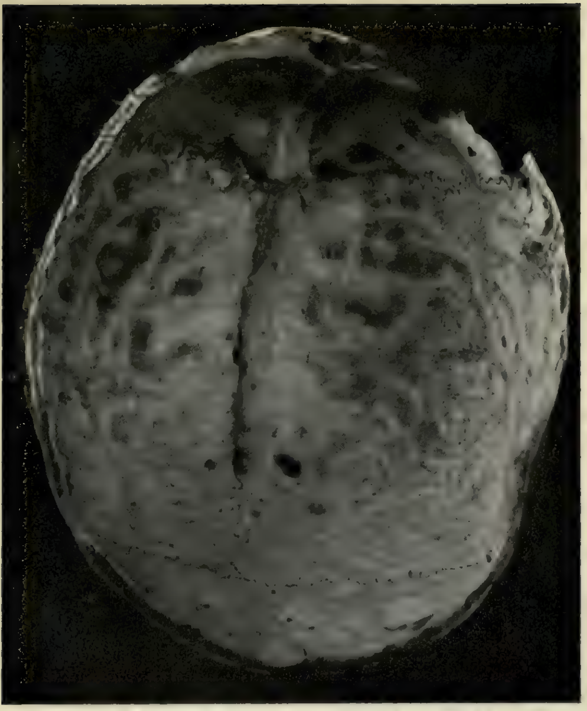

Fig. 177.—Glioma of cerebellum. Calvarium from within, showing the deep markings of the brain convolutions in the bone. The skull-cap was very thin, and in places translucent.

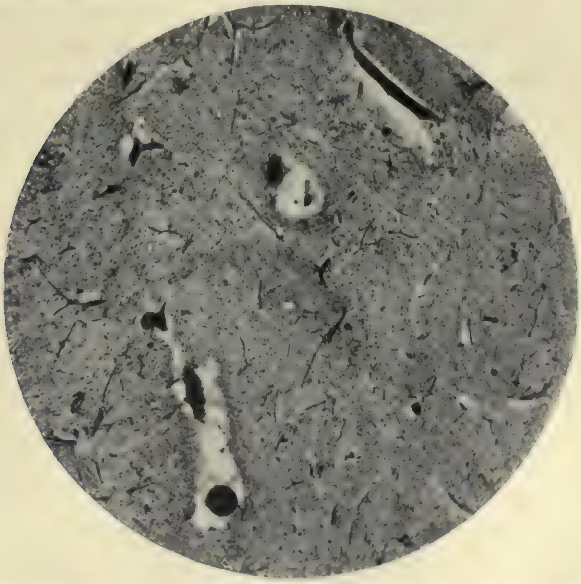

Fig. 178.—Photomicrograph (low power) of section showing glioma of cerebellum with psammoma bodies. The cell-outlines do not show in this specimen, but the general appearance of the tumor section is very characteristic.

similar to that which was found in the fourth ventricle when the latter was opened and is probably the result of the degenerated tuberculoma that produced the intracranial pressure in the first condition. On further sectioning of the right cerebellum two more tumors were found, one 15 by 7 mm. and the other one 5 mm. in diameter. These were in structure like the anterior end of the olive-shaped tumor previously described and were red.

The calvarium shows a great atrophy of the inner plate. The osteoporosis is so extensive that at places the calvarium is translucent. A microscopic examination made of the tumor mass showed the tumor to be gliomatous in structure. The degenerated area was found to contain lime deposits of the psammoma type. The pathologic diagnosis is as follows:

1. A glioma of the right cerebellar lobe, with large apoplectiform cyst and a necrotic area with lime deposits of the psammoma type. 2. A moderate hydrocephalus internus. 3. Marked edema of the brain. 4. The large cyst in the cerebellum was either a primary cyst, or resulted from the liquefaction of a tuberculoma that was probably the cause of the pressure before the primary decompression was done, six and one-half years ago. The autopsy showed that it was impossible to do any successful operation for the removal of the tumor. 5. Extensive osteoporosis of the inner plate of the whole calvarium.

SURGICAL CLINICS
OF
JOHN B. MURPHY
Volume II Number 5

DOUBLE INGUINAL HERNIA.—SOME ITALIAN STATISTICS.—TECHNIC OF THE ANDREWS OPERATION

HISTORY

Patient, male, aged fifty-three, married, enters hospital because of a swelling in the right inguinal region. He has no pain, but a mass pushes down into his scrotum whenever he removes the truss he wears. The hernia was never strangulated. His doctor told him that he also had a small hernia on the left side. The trouble began about twenty-five years ago, when he strained himself in a boat-race. Soon afterward he noticed a small swelling in the right inguinal region. He began wearing a truss at once, and continued to wear it all the time during next six or eight years. Then the hernia disappeared, and he did not wear a truss from 1898 until three years ago, when the swelling reappeared in the same place. He again began wearing a truss at once, but the swelling enlarged gradually, and now the mass descends into the scrotum when the truss is removed. It can easily be replaced in the abdomen by manipulation. The abdomen bulges in the right inguinal region on coughing. When he is lying down and quiet there is no bulging. For the past year the truss has not kept all the mass in the abdomen—a small mass slips down from under the truss into the upper part of the scrotum.

Past History.—He had pneumonia when fourteen years old; was sick three weeks. He has had several attacks of grip and tonsillitis. Five weeks ago he slept out-of-doors, and the next morning he had pain and stiffness in the back, chest, and neck; in fact, he "felt stiff" all over. He still has stiffness and pain in the neck. He had syphilis in 1881, for which he took treatment for two years. He had a Neisserian infection in 1885. Father, grandmother, four uncles, and an aunt died of apoplexy between sixty and seventy years of age.

COMMENTS AND OPERATION

Dr. Murphy: That is a typical history. Inguinal hernia constitutes about 80 per cent. of all hernias, and occurs more commonly in males, more often on the right side than on the left, owing to the later descent of the right testis, which keeps the right inguinal canal patent for a longer period. A good classification of inguinal hernia is the one given on the board.

- I. Indirect or oblique
  - Acquired
    - Incomplete.
    - Complete (scrotal or labial).
  - Congenital
    - Vaginal.
    - Funicular.
    - Infantile.
    - Encysted infantile.
- II. Direct (always acquired).
- III. Interstitial (usually congenital)
  - Intraparietal
    - Intra-iliac.
    - Antevesical.
  - Interparietal.
  - Extraparietal.

The indication for treatment is clearly a surgical procedure. We have been doing herniotomies for this type of hernia in a typical way for a number of years, namely, making a straight incision through the skin high (about one inch) above and parallel with Poupart's ligament, extending from the external to the internal ring, and *down to the aponeurosis of the external oblique muscle, well above the canal, and never on the elevated ridge of the hernia.* The ilio-inguinal nerve and epigastric vessels are searched for and retracted out of the field of operation. That gives us a very good exposure of the canal and its contents. By this procedure we are never at a loss to know the location of any of the structures here. Next we divide the aponeurosis of the external oblique muscle high up—the highest portion of the hernial tract and of the inguinal tract. By doing that we can make a good canopy for the new canal.

The Andrews operation is the operation of election in these cases. The Andrews and the **Bassini** operations are the two which we perform. We do them with great regularity and we are well satisfied with the results. The next step is to bring out of this inguinal canal everything that is in it, so that you are clear to the margin of the rectus muscle. We elevate the contents of the canal and retract all these structures upward and outward by passing a piece of gauze under them. That brings the conjoined tendon clearly into view. The cremasteric muscle and fascia are raised from the cord, divided between forceps, and ligated. Next we expose the sac. The sac should be on the upper and inner side of the cord. We will isolate it by gentle gauze dissection so as not to injure the vas deferens, which is easily located by running the cord through the fingers and spreading out the tissues. We have no difficulty in locating this sac. We open it with the dissecting scissors and catch the edges with hemostats. Now we will return the contents of the sac into the abdomen. It is a small mass of omentum, and it is irreducible. It is adherent to the sac-wall. We will enlarge the opening in the wall of the sac a little, so that we can ascertain where the omentum is fixed. It is fixed away down in the end of the sac. We will ligate a little point of omentum off where it is adherent to the sac and return the proximal portion into the abdomen. Now we have the sac clear. The next step is to separate the sac from the vas and the vessels. That is done with a sponge. Put your finger into the sac and slowly push the external tissues downward and away from the sac with a gauze sponge, which, you see, I am accomplishing here. The most adherent tissue will be the vas. Now that that is accomplished, we are ready to close the sac by means of a ligature and a suture. There is rarely ever any danger of ligating anything in the sac, because you can see into the sac, but there is often danger in ligating something outside the sac; the most common tissue is the bladder. Therefore we are very careful to locate the bladder before we tie the ligature around the neck of the sac. A second structure which may be ligated is the vas. We locate the vas. It is out of the way, and we are ready to tie the ligature, which consists of plain No. 2 catgut.

The next step is to sew that sac, making it a part of this ligature. One end of the ligature is threaded in a needle, and I put in a puckering string on the inside of the sac external to the ligature. When I tie that suture the sac will be made a part of the ligature and it cannot possibly slip out. You have it not only ligated, but you have it ligated so that it cannot escape.

What disposition will we make of the remaining portion of the sac? Do we remove the sac down in the scrotum? Not ordinarily. Why should we traumatize the scrotum? In this case, however, we will remove the sac because it has not passed down into the scrotum for more than half an inch. There is no trouble in removing all that sac without inflicting the least trauma on the scrotum; if it extended down into the scrotum for any considerable distance, we would not endeavor to remove it, for the reason stated. The next step is to reduce the bulk of tissue in this cord by gently pushing it downward through the external opening or ring of the inguinal canal. There are the vessels and there is the vas. You can see it slipping between my fingers. We can remove a large portion of this cremasteric muscle and fat. We put on two hemostats, one above and one below, resect the tissue between them, and ligate the stumps with catgut. We will push one end down and the other end up. We are now ready to close the canal. We have taken out the sac and we have reduced the bulk of material in the canal by fully one-third. Now we have only a small cord. The next step is to close the canal. We will make a union of the upper edge of Poupart's ligament to the conjoined tendon and to the aponeurosis of the external oblique. Here is Poupart's ligament. Remember, we divided the aponeurosis of the external oblique high up over the canal, so that we could get a strong flap for the new roof we are going to make for it. The Andrews imbrication will now be made. Catgut is used to suture the flap. Raising up the cord, we get a good exposure of the field. We place these stitches in regular order, beginning above. We first catch up the thickened portion of Poupart's ligament, then pick up the conjoined tendon and the external oblique, passing the needle under the cord and then back again to Poupart's ligament (as indicated in Fig. 189

and subsequent ones kindly furnished me by Dr. E. Wyllys Andrews). When that stitch is tightened, the upper flap is imbricated under the cord and forms the floor of the new canal. The lower or top flap, consisting of the external oblique aponeurosis, will form the roof, passing over the cord. That is stitch No. 1. Then comes stitch No. 2; we pick up a portion of the internal oblique and the aponeurosis of the external oblique. That is the great advantage of the Andrews operation in that it utilizes the aponeurosis of the external oblique twice. We insert as many of these sutures as are necessary—usually four or five.

I am using a double chromic gut suture. We always prefer a double suture to a single one of larger size, because it is so much easier to sterilize a smaller diameter gut than a single heavy strand. The last stitch at the lower angle makes the strongest support. That is in the line of the vas and the vessels, picking up the internal oblique and transversalis and again picking up the aponeurosis of the external oblique. All the knots will be below the cord and under Poupart's ligament.

Now we are ready to tie these sutures and complete this portion of the operation. We start with the upper. As we draw the suture taut you will note how the upper flap is drawn down under the cord to Poupart's ligament and what a firm support it gives to the canal. After having tied all these sutures, the next step is to drop the cord back into the scrotum and to suture the canopy. The lower flap is drawn over the cord and sutured to the upper portion of the external oblique aponeurosis. That gives us practically a new canal, and it has firm walls, so that there is little chance for a recurrence of the hernia.

That completes the operation, except for the closure of the skin incision, which is done with horsehair. The hernia on the opposite side will be treated in the same way. The sac on this side extends only into the canal. It is not in the scrotum at all.

Let the record show that Sister reports the sponges counted and correct. Let the record further show that on the right side the sac extended a short distance into the scrotum; that in the left side it came down only to the external opening of the canal.

On the right side the sac was embedded and sutured after a careful inspection of the inner and outer surfaces of it to assure us that no other tissue was ligated. Remember, the bladder was exposed in both hernial canals, and if it was not for my precaution of never ligating tissue outside of the canal, I would have had a ligation of the bladder outside of the sac, and I cite these illustrations to call attention to the danger. The caput coli has been sutured and cut off in the sac; the ovary has been cut off and not recognized until subsequently; the appendix has been cut off in the sac. The bladder has been included in the ligature more frequently than any other structure, because it is not in the sac—it is on the sac.

We use horsehair for closing the skin incision because it has an advantage over silkworm-gut in that it does not produce a necrosis. Silkworm-gut is like wire—it is inelastic and produces a necrosis if it is tied tight. So far as capillarity is concerned, it has no advantage.

These wounds will be sealed with collodion gauze. No other dressing will be applied.

It is interesting to examine the results of Dr. G. Fantino's experience in 4580 cases of hernia which he operated. Full details of only the first 4200 are given, as the remaining number were too recent to be available for percentage purposes. There were 89.4 per cent. of inguinal, 7 per cent. femoral, and 3 per cent. umbilical hernias. Males furnished 86.45 per cent., and females, 13.55 per cent.; inguinal hernia in males, 93.8 per cent.; femoral, in females, 71.4 per cent.; umbilical, in females, 82.7 per cent.

This is a large number of cases in the practice of one man, and must represent fairly well the percentage proportion of hernia in Italy, although it may not accurately represent the percentage proportion for other countries, as the relation of women and men respectively to labor and laborious occupations differs in Italy from that of other countries.

Strangulation was found in 6.5 per cent. of the inguinal hernias, 10 per cent. of the umbilical, and 38 per cent. of the femoral. This throws an additional light on the danger of femoral hernias, and impresses on one the importance of an early radical operation for its cure. When one thinks that 4 out of 10 of all the cases that came to Fantino were strangulated, it shows the urgency of the early radical operation for its cure. The proportion of strangulated hernia in females was a striking one. While it will be noted that the males represented 86.5 per cent. of all cases, there were only 251 strangulations; and the females, representing 15.3 per cent., had 124 strangulations, or almost one-half as many as that of the great number of males.

Fantino used iodized catgut, as a rule, in his operation, and finally, after a trial of the many operations for hernia in vogue, adopted the Bassini as the ideal one. Among his 4200 cases there were 9 fatal issues, one each from infection, pulmonary embolism, and hemophilia, and six from chloroform intoxication. This latter is an appalling statement, as it shows that the deaths from chloroform intoxication were greater than from all other causes combined, and one can scarcely understand how chloroform can be continued as an anesthetic with this condemning conclusion.

Recurrence was observed in 74 cases: before the age of twenty, 1 per cent.; between the ages of twenty and sixty, 4 per cent.; and over sixty, 9 per cent. Twenty-seven of the 74 recurrences took place in the direct hernias, showing that some improvement must be made in the management of direct hernias, and it appears to us that the Andrews operation does not suffer from this defect as does the Bassini. Sixty-five per cent. of the recurrences occurred in the first year and 25 per cent. in the second. Therefore after the second year there is 10 per cent. of chance of recurrence with the Bassini operation.

Polya believes that an indispensable part of the operation in order to avoid recurrence should be the fixation of the aponeurosis of the rectus to Poupart's ligament. This is a rather difficult procedure, unless the sheath of the rectus is split. He does the Bassini operation. With the Andrews operation the suturing of the rectus muscle to Poupart's ligament is not at all an important factor.

It is difficult, if not impossible, for us in Chicago to make an accurate analysis of the final results of our work, as the population is so migratory that it is not possible to obtain reports on anything like a considerable percentage of cases subsequent to operation.

The proportion of left and right inguinal hernias can well be judged by Balfour's paper from the Mayo clinics. Of 1889 operations, 795 were right and 466 were left inguinal hernias. He shows also that the McBurney operation carries with it a risk of right inguinal hernia through interference either with the muscle or the nerve supply of the right inguinal tract. He attributes the larger row to the right.

The iodized or chromicized catgut has become almost the uniform suture material in hernia in America. There are a few faddists who still use wire and silk, and who pay the price of either removing the suture material themselves or of having their colleagues in some distant part of the country take it out for them. It is a well-recognized fact that silk of a considerable size may remain inert for years,—eight, ten, twelve, and more,—and finally suppurate and have to be removed. The explanations given for this phenomenon are not quite satisfactory.

[Wounds healed per primam. Stitches removed on eleventh day. Subsequent progress of case uneventful.—Ed.]

TECHNIC OF THE ANDREWS OPERATION FOR HERNIA

Edited by Dr. E. Wyllys Andrews for this Number

This herniotomy is done in fifteen or twenty minutes in simple cases. Two types of imbrication or overlapping—a posterior, behind the cord, and an anterior, in front of the cord—I described in the original publication of eighteen years ago (Chicago Medical Recorder, July, 1895). One of these has since been printed as the "Girard operation," and one as the "Hopkins operation," with the statement: "This is called the Andrews operation, but we devised it independently."

Posterior imbrication appeals to those who like the Bassini method; anterior imbrication, to those who object to "transplanting the cord." It leaves the cord behind the deep suture line. There is a tendency of late to do more anterior operations, in which Dr. Coley, of New York, informs us that he uses both types, but rather more often the old-fashioned posterior method.

The minor details described below may vary with different operators, but should never vary with the same operator. Speed and smoothness come from uniformity, followed for uniformity's sake. Each operation becomes a rehearsal for the next.

Step 1: Skin Incision.—Location, 2 or 3 cm. above Poupart's ligament, parallel to its inner two-thirds. Length, 15 to 18 cm. in adults. Pinch up a fold of skin transverse to the groin with the left hand, the assistant holding the same fold 3 or 4 cm. distant. Raise this fold 7 to 10 cm., and cut its whole height by one stroke of a large keen scalpel. Continue holding the pinched-up margins, relaxing slightly until the epigastric and other vessels are seen and secured. Then drop the skin and pick up every smallest bleeding point. Do not allow even droplets of blood to continue running. Expose the external ring and aponeurosis cleanly.

Step 2: Opening the Canal.—Do not insert a director and split the ring up from below, but split the external oblique aponeurosis the length of the canal by one straight cut from above between its fibers. Evert the lower segment and run the scalpel handle along its inner aspect, exposing freely the shelving inner edge of Poupart's ligament.

Step 3: Removing the Sac.—Either lift up the cord mass on the finger, which requires some blunt dissection, or let it lie adherent, according as you wish to repair the ring behind or in front of the cord. In either case remove any lipomatous masses high up by tying off above the ring. Do not remove any veins or cremaster strings. Cut a button-hole with a small, keen knife in the fascia propria of the cord until, at just the right depth, you see the pearly-white sac-wall. It is like a hydrocele dissection. Seize this wall with blunt forceps, and strip it rapidly out by sponging away the cord. Follow well up into the abdomen, and a place will be found where the cord and sac diverge, so that the sac-neck can be ligated without inclosing the vas or vessels.

In scrotal hernias strip out the sac if easily separable. If not, cut off the part in the canal, and return the scrotal part to the scrotum, either with a gauze drain through the lower end of scrotal skin or else everted, like a hydrocele operation. Pull the testes freely up into the wound whenever necessary. In several cases the scrotal sac formed a hydrocele requiring subsequent tapping, but in many others caused no trouble. Separate the parietal peritoneum 2 or 3 cm. all around the ring with the finger, so that it is loose from the muscles and can be drawn down freely. Open the sac widely at its fundus, and look very carefully all over its interior for adherent omentum or bowel, especially at its neck. Keep this opening under the eye up to the moment of ligating, and watch carefully that nothing slips into it. Simple ligation for narrow sacs, and suture ligation or purse-string closure for large ones, are best. The Macewen pad of the folded-up sac has no great faults or merits. Andrews sometimes uses it in a typical large hernia to increase for a time the amount of new deposit at the weak point, but is somewhat skeptical of its permanent value. On account of the loosening of the surrounding peritoneum, the cut-off sac will retract well beyond the muscles. If the stump lies visible in the ring, it is too long, and will form a funnel or dimple inside, tending to bring on another protrusion. If the stump does not disappear wholly, remove more of it.

Step 4: Deep Suture.—(*a*) *Anterior Imbrication.*—Place from two to five strong stitches uniting the conjoined tendon and muscles together with the upper segment of external oblique firmly down to Poupart's ligament, all in front of the cord, which is thus pushed backward and toward the pubes. The lower flap of external oblique is left free for step 5.

(*b*) *Posterior Imbrication.*—Lift up the cord on a loop of tape or Andrews' hernia-retractor, which holds the wound open and has an adjustable hook to suspend the cord. Pass the deep stitch behind the cord by first lifting Poupart's ligament on the left forefinger and pushing the needle from outside the fascia behind it. The needle-point is guided by the forefinger and cannot injure the vessels. It is vitally important in direct hernias

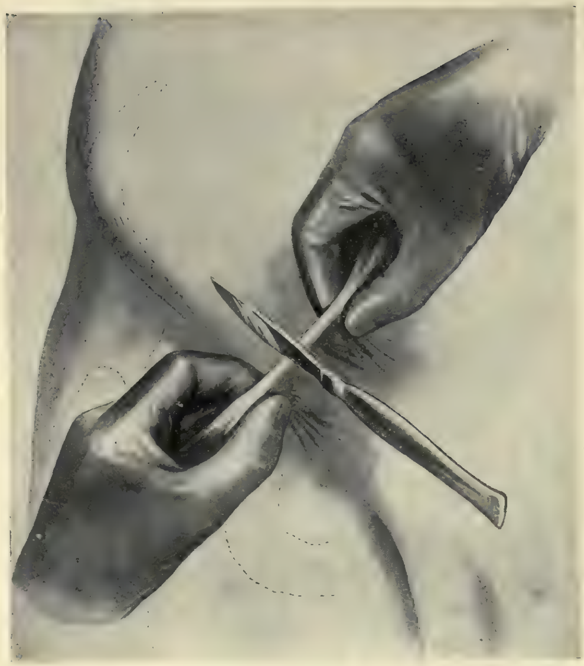

Fig. 179.—The Andrews operation. Step 1: The skin is pinched up while the incision is being made (Bryant and Buck).

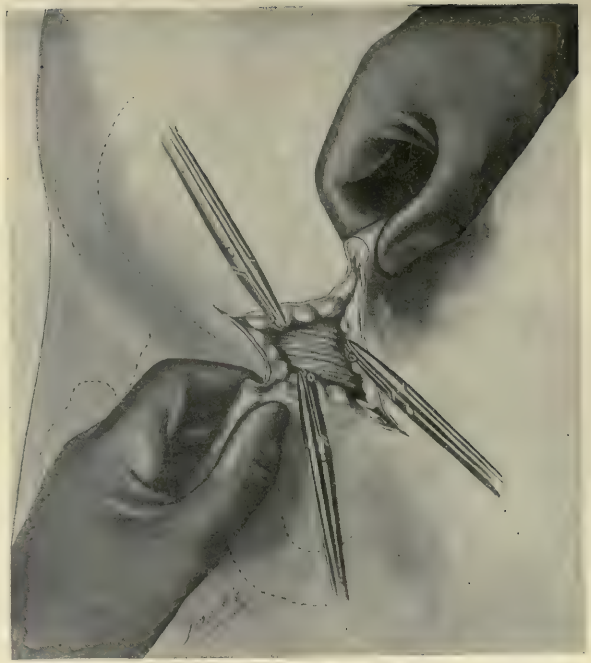

Fig. 180.—The Andrews operation. Step 1 (continued): The skin-flaps are held while the artery forceps are applied one after the other (Bryant and Buck).

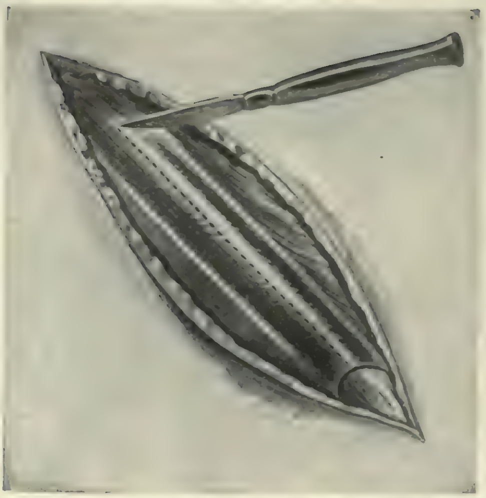

Fig. 181.—The Andrews operation. Step 2: Dividing the external oblique (Bryant and Buck).

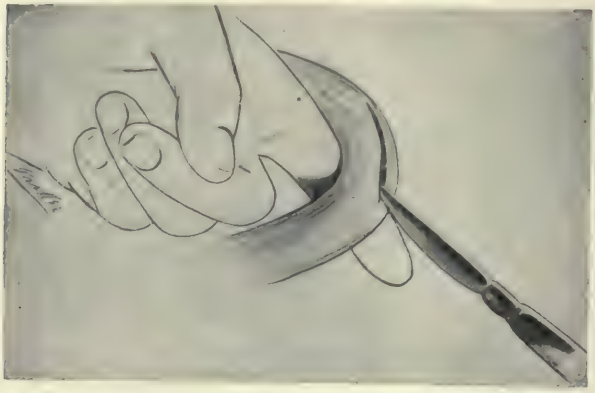

Fig. 182.—The Andrews operation. Step 3: Dividing the cord layers in order to find the sac (Bryant and Buck).

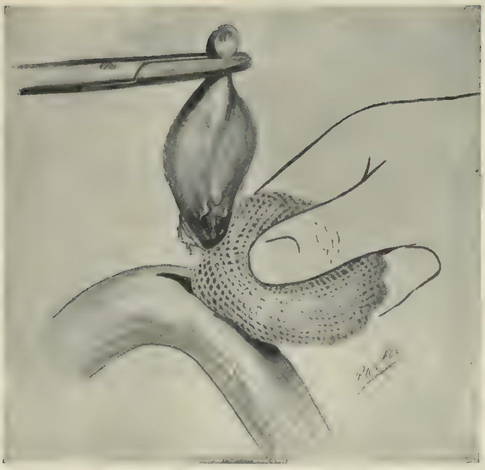

Fig. 183.—The Andrews operation. Step 3 (continued): Gauze sponge used for stripping the coverings from the sac (Bryant and Buck).

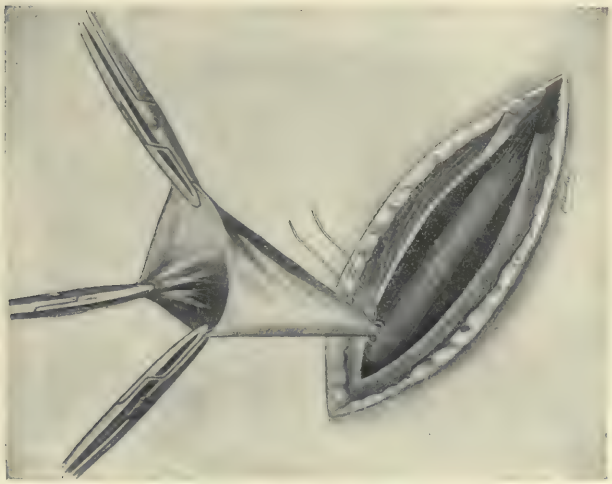

Fig. 184.—The Andrews operation. Further continuation of step 3: Sac deligated. The fundus is open for inspection (Bryant and Buck).

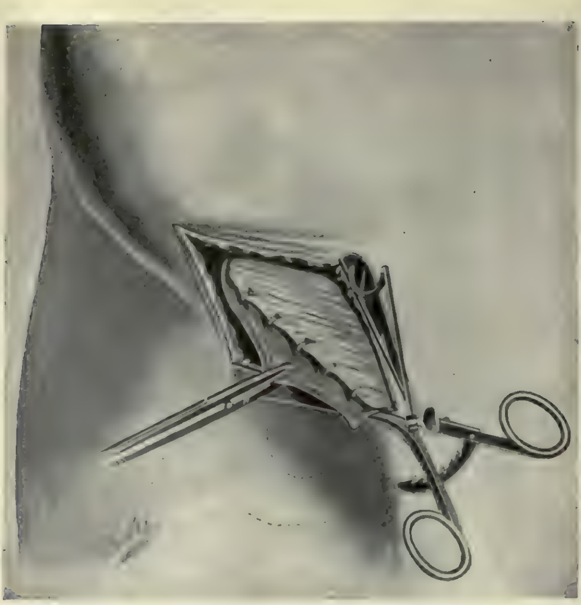

Fig. 185.—The Andrews operation. Step 4: Anterior imbrication (Bryant and Buck).

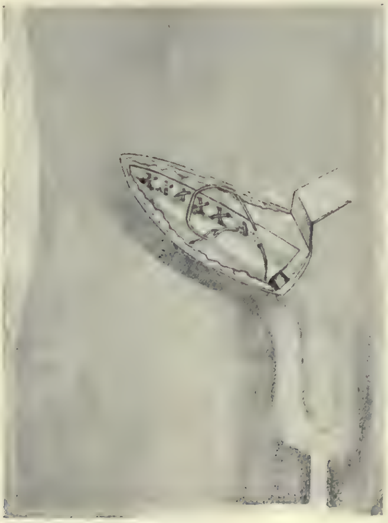

Fig. 186.—The Bassini-Fournel operation. The drawing shows the external oblique aponeurosis overlapped (from Bouveret). Another method of holding overlapped flap in connection with Step 5 (Bryant and Buck).

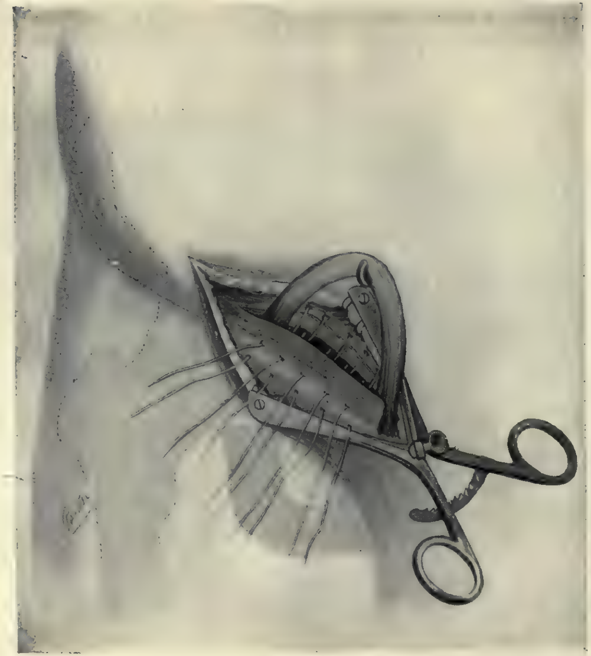

Fig. 187.—The Andrews operation. The drawing shows the manner of using the hernia-retractor in posterior imbrication (Bryant and Buck).

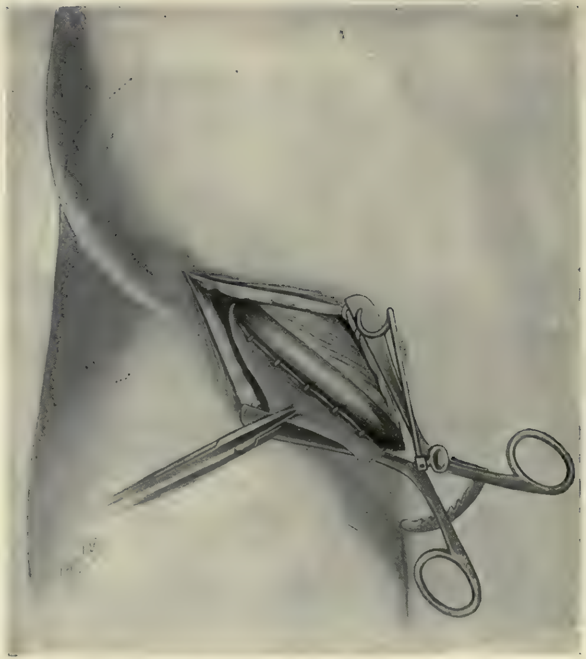

Fig. 188.—The Andrews operation. The cord is here shown to have been restored to its natural position (Bryant and Buck).

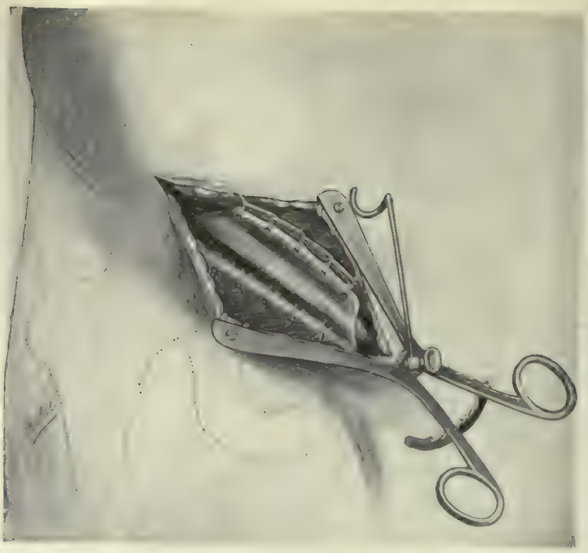

Fig. 189.—The Andrews operation. Step 5: Second buried suture; layers overlapped. Note the close resemblance of the Fournel method to this procedure (Bryant and Buck).

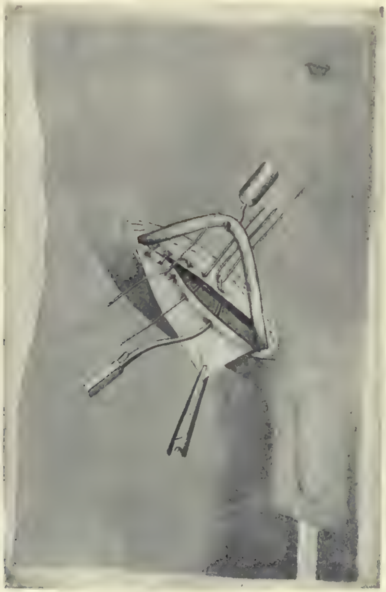

Fig. 190.—The Bassini-Fournel operation, identical with the Girard and the Andrews operations (Bryant and Buck).

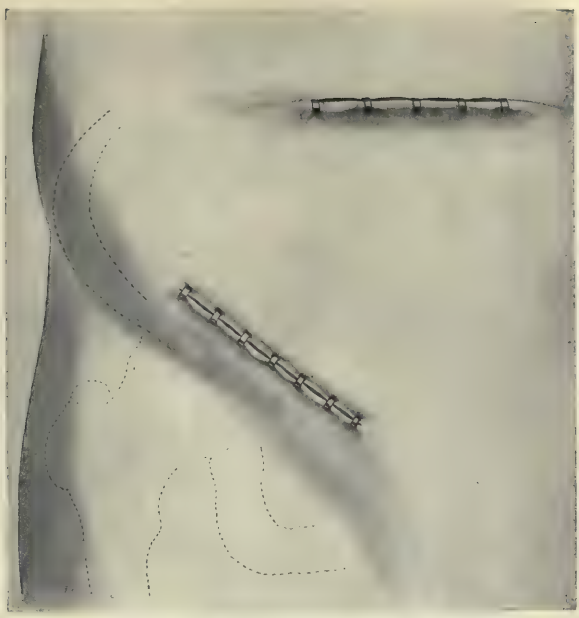

Fig. 191.—Step 6: The external wound closed by metal clips, which give a ridge-shaped suture line (Bryant and Buck).

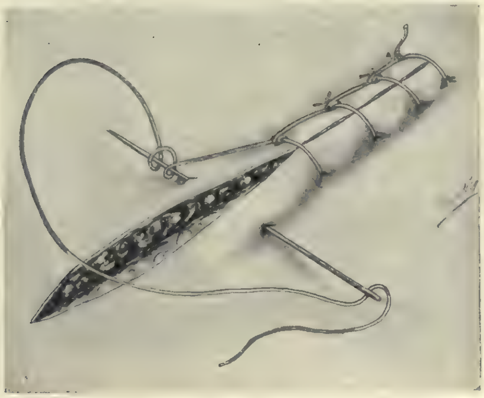

Fig. 192.—The Andrews operation. Step 6: Method of closing the skin wound by double-locked buttonhole stitches. Note the fact that the slack thread is wound twice around the trocar needle-point, causing each segment to remain taut like an interrupted suture (Bryant and Buck).

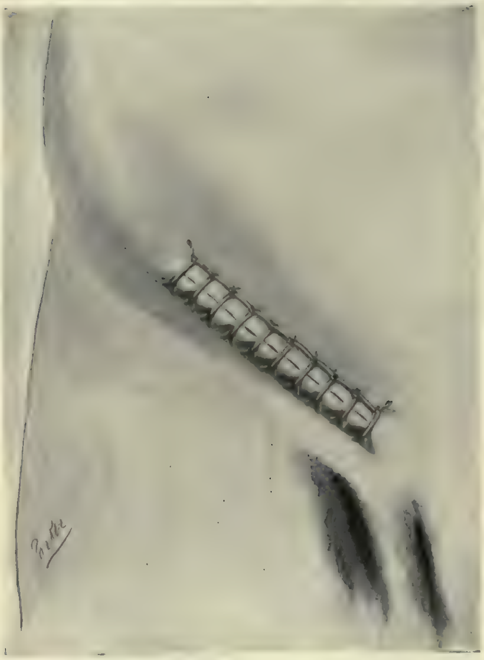

Fig. 193.—The Andrews operation completed. The skin remains in a ridge-shaped line like that which results from the use of the clips. If plain catgut is used, this ridge flattens out in from five to seven days (Bryant and Buck).

and large rings to get the first deep stitch as near the pubic end of the ligament as possible. It often includes a little of Gimbernat's ligament. From its entrance point the needle is guided by the forefinger into the canal. It is then picked up and the opposing muscle entered from within outward. The lowest or first stitch in large rings should include a little of the rectus muscle, the sheath being split open to allow this. In small rings this is not necessary. Do not resort to the Wölfler method of everting a trap-door flap of rectus sheath or any other mutilating flap methods, except in very large or atypical hernias or those in which former operations have destroyed the usual landmarks. The imbrication is of itself a perfect flap transplantation, and will fill the largest rings without great tension. After transfixing the transversalis fascia and conjoined tendon or muscles, we have what would be a Bassini stitch in mattress form. To this we now add the external oblique margin, either in the same loop or by passing the needle, which has already pierced the deep muscle, down and forward through Poupart's ligament and back to catch the external oblique. The needle is next passed out just in front of Poupart's ligament, so that it emerges about 5 mm. in front of where it entered, and surrounds the ligament. It is thus a mattress stitch, with the knot just inside the skin and fat of the groin. From two to five such deep stitches are used. It is a mistake to place them very near together or tie very tightly. The uppermost or outermost stitch should narrow the ring snugly around the cord. Tie this one first and watch the effect on the cord. Commonly, its artery can be felt pulsating before and after tying. If in doubt, discard the upper stitch.

Step 5: Second Deep Suture.—Lap the lower segment of external oblique across the upper. In case the cord has been raised, it goes between these layers. They overlap about 2 cm. at the external ring, thus strengthening and narrowing this dilated orifice. Suture them in this position tight enough to bear part of the strain on the deeper stitches by a lock-stitch running seam. It will then be seen that the strength of the abdominal wall has been doubled by redoubling its thickness; also that these overlapped surfaces are more sure to unite firmly than in the edge-to-edge of other methods.

Step 6: Skin Closure.—Andrews prefers metal clips, but if desired, after very thorough hemostasis, sew the skin with a trocar-pointed needle held in the fingers. The following form of buttonhole continuous suture will please those who have not seen it.

After the first knot, thrust the needle through both flaps about 1 cm. from their edges. With the skin thus transfixed wind the slack of the thread twice around the point, instead of once, as in the ordinary lock-stitch. Then draw it up until it is just tight between the stitches but does not pucker.

There is no back-slip, and each stitch holds by friction. This gives a ridge-shaped suture-line, like a double row of stitches. Why it does so one cannot explain, but no other suture looks like it.

Do not drain these wounds, except in rare cases, where there is much dissection and persistent oozing, when it is well to insert a small drain through an independent buttonhole—not through the angle of the incision.

Seal the wound with collodion. Firmly bandage both groins with wide spica bandages. In children and restless patients it is sometimes well to pad the hips, trunk, and thighs with antiseptic cotton, and put a light cast or starch bandage from the chest to the knees.

There is no advantage in redressing these cases. They are well on the eighth or tenth day, and should be allowed to be up and take ordinary exercise at once. Let no binder or truss be used after discharging the patient.

APPENDICITIS—DIFFERENTIAL DIAGNOSIS; PERFORATIONS.—TREATMENT OF GENERAL SUPPURATIVE PERITONITIS

HISTORY

Patient, female, aged eighteen, single, enters hospital complaining of constant pain and slight tenderness in the right iliac fossa. No fever or vomiting. The trouble started seven years ago, when she first began to have a dull pain in the right iliac fossa. This pain has been present about half the time, with daily severe exacerbations in the same region. The pain disappears and returns two or three times during the day, and is often very severe for two or three hours at a time. She says that there have been very few days since the trouble began that she has not had one or more attacks of acute pain in the right iliac fossa, each lasting one to two hours, the pain then becoming dull in character. It has always been localized, and bears no relationship either to eating, kind of food eaten, nor menstrual period. She does not think that she ever had fever. She has never vomited, but has had occasional attacks of nausea with pain all over the abdomen. When the sharp pain comes on she has to loosen her corset to get relief. She has had a little tenderness over the right iliac fossa continuously during the past year, and she thinks that the pain has been increasing, both in severity and frequency, since a year ago. During the past month she has not been able to sleep much, because of the attacks of pain at night. About a week ago the pain became so severe and the attacks recurred at such short intervals that she consulted a doctor. At this time she was tender over the appendiceal region. She has no temperature elevation now. She had scarlet fever when eight years old; was sick six weeks. When twelve years old she was in the hospital three months; was not very sick, and does not know what the trouble was. Menstruation began at sixteen; regular twenty-eight-day type; flows two days; one napkin a day. Last period a week ago. Family history negative to tuberculosis.

COMMENTS AND OPERATION

DR. MURPHY (Operation June 26, 1913): This is one of the atypical cases of appendicitis. She has a leukocyte count of 11,600. It differs from two cases we had yesterday. Each of these two patients had at one time—one as long as seven years ago—a typical acute attack of appendicitis, but no subsequent acute attack. Occasionally the patient had mild attacks of discomfort and pain. Notwithstanding the atypical history we found in each case gross evidence of a pathologic lesion.

We now find it difficult to keep from operating for appendicitis. People have appendicitis on the brain. They want the appendix removed for almost every pain they have in the right side. One has to keep constantly alert to keep from making errors. One lesion you are most likely to confound this chronic type of appendicitis with in the early stages is what? Ureteral stone or renal stone. The patient before us today has no blood or pus in the urine. She has nothing to indicate a stone in the kidney or ureter. One is also likely to confound appendicitis with tuberculosis of the tubes in girls. Remember, tuberculosis of the tubes occurs more commonly in girls than it does in women. It has a distinct periodicity, but the real manifestations of the attack are more pronounced than they are here. It commences and progresses just like an acute attack of appendicitis; the patient has pain, nausea and vomiting, local sensitiveness, elevation of temperature, but no pronounced leukocytosis. In this case there is, without the other symptoms being present, a pronounced leukocytosis of 11,600. We do not make a differential count, because we do not believe that the polynuclears have any important relation to the diagnosis. We do believe, however, that they bear a relation to the virulence of the infection and to the resistance response of the patient against infection, but we rely more on the total number of leukocytes in making a diagnosis of some inflammatory or infected lesion than on the differential leukocytic count. The cases in which leukocytosis is of most value are the acute ones. We have had only 5 cases of acute appendicitis in which there was no elevation of temperature. One of these patients had a moderate leukocytosis; the other had an acute appendicitis with a perforation into the free peritoneal cavity, with neither a temperature elevation nor leukocytosis. That patient was right here in the hospital and under observation all the time. He was being treated for another condition—an arthritis—from which he was convalescing, *and was receiving vaccine injections every second day*. It was the scaphoid abdomen that led us to operate on him. He had nausea and vomiting, local sensitiveness and pain, but he did not have the temperature elevation nor the leukocytosis. Why? He was receiving large doses of an autogenous vaccine for his arthritis, and we believe that kept him from having rise of temperature and a leukocytosis. That misled us in the diagnosis. That is the only case in which we have had an absence of both leukocytosis and temperature elevation. The other patients in which the temperature rise was absent had a leukocytosis of 11,000 or 12,000. That makes only about five cases that we have had without temperature elevation, or about one to every 2000 cases that we have had. That is a very low percentage. A very instructive and interesting case that came under my observation was that of a doctor's daughter I was called to see one Sunday evening many years ago. In the forenoon she had had severe pain in the right side of the abdomen, with nausea and vomiting. She had the local sensitiveness, but she had no temperature elevation. Her father said he took the temperature regularly and always found it was normal. I felt that perhaps he had not taken the record as accurately as he should, and on the strength of the other symptoms I made a diagnosis of appendicitis. We took the patient to the Presbyterian Hospital and got her ready for operation. She was anesthetized at once, but I was so impressed with the fact that she had no temperature that even after I had raised the scalpel to make the incision I laid it back again and palpated the point of resistance. I was suspicious because of the absence of temperature. Suddenly the tumor which I had detected preceding the anesthesia and which was so sensitive disappeared upward on palpation under the right costal arch. It was the kidney that had been displaced. The appendix was not at fault at all. I did not operate. When she came out of the anesthesia she told this story: She was at a picnic two days before and was leaning up against a board fence which was just high enough to catch her under her costal arch. A young man took hold of her feet, pretending to throw her backward over the fence. She turned to the right, the top fence board caught her kidney and tore it loose, pushing it down into a subperitoneal position at McBurney's point. We did not open the abdomen, for which we, of course, were mighty happy.

It was a classic history of appendicitis, except for the absence of temperature elevation. That shows you how much stress I place on the element of temperature elevation.

We insist on elevation of temperature as a symptom some time during an acute attack. When there is no temperature elevation in the middle of the attack, it means that there is no continued absorption of toxic products from the appendix, but in all these cases there is a rise of temperature in the beginning. It may escape detection, but it is there just the same. I place great stress always on these five symptoms—pain, nausea and vomiting, which is always a reflex symptom, the local sensitiveness, which is due to two things,—the infection and the tension under which the products of infection are held,—the elevation of temperature, and the leukocytosis. Originally we considered only the first four symptoms. We have added the fifth—the leukocytosis, which is at times a very important factor in the differential diagnosis. We believe that if these symptoms occur in that order the case is one of appendicitis. When the temperature comes first, or if the nausea and vomiting come first, we always question the diagnosis.

The lesions that are most commonly confounded with appendicitis are, first, renal calculus, and, second, cholelithiasis. In an acute attack you rarely will make a mistake if you remember that there are a few essential and positive signs that are always present in these three lesions. First, pain is always present in the three; second, nausea and vomiting are commonly present in all three because they are reflex symptoms; third, the local sensitiveness is in a different place in each one of the three; fourth, the elevation of temperature is not present in a cholelithiasis unless sepsis is associated with it; fifth, the leukocytosis is not present in any of the three unless infection is present with it. But you have definite differential physical signs always, easily determinable at the bedside. What are they? First, the sign of sensitiveness. What is that due to? When you have a gall-bladder distended with fluid it is just like a distended urinary bladder. Hook your finger under the right costal arch, and ask the patient to take a full breath so that the gall-bladder is pushed down below the arch; then strike the finger forcibly with your other hand, making a sudden strong downward pressure. If it is an acute gall-bladder lesion, he has pain so severe that he feels like resenting the injury by physical violence. That is a positive sign. If it is an appendix lesion, you will find that you are able to press your left hand deep into the left iliac fossa, after you have gained the confidence of the patient and he relaxes enough, but he will not let you press your hand in the right iliac fossa. Finally, in cases of renal calculus with a distended kidney pelvis, there is just the same tension in the pelvis of the kidney as there is in the case of the distended gall-bladder. The method we originated for use in such cases is this: Get the patient to the side of the bed, have him lean forward and drop his arms down so as to arch his back. Let us presume the lesion is in the right kidney. If it is, he will be sensitive on the right side. Give him a good blow with the closed right hand on the left hand placed firmly over the left kidney first and he will give no response. Then you do the same over the right kidney and he straightens up suddenly and cries out with pain.

I like to quote the case of a lawyer who had a stone in the ureter who was sent to me for operation for acute appendicitis. I gave him that test. The patient was of Celtic stock, and he said: "Doctor, it is all I can do to keep from hitting you; you hurt me so and you did it intentionally." We examined his urine immediately and found blood in it. In four or five days he passed his renal calculus and we did not have the humiliation of opening the abdomen for an appendicitis when the lesion was in the kidney. This fist percussion (Murphy test) is one of the most positive tests in surgery. It is present with a renal calculus; it is present with an acute infection of the kidney. Why? Because the whole pelvis is filled with infective products under tension and the tension is what makes these patients so sensitive to percussion at the bedside. You can make a differential diagnosis in the first hours of an appendicitis, a gall-bladder lesion or a kidney lesion better than at any other time.

It is unnecessary for me to speak of the desirability of early operations—that point is conceded by everybody. It is conceded that no man, no matter how large his experience may be, is able to judge from the early symptoms whether the case is going toward a favorable or an unfavorable result; in other words, in the first twenty-four to thirty-six hours you cannot tell from the symptoms whether the case is going to terminate favorably or unfavorably, from a pathologic standpoint. There is just one thing to do and that is to take out the appendix while the infection is still confined within its walls. I think the expression which I used at the International Congress at Budapest tells the story: "When an abdomen is opened and shows pus outside of the appendix, as a result of a perforation, its presence is an incontrovertible proof that the case has been badly managed up to that time." Explanations do not help to relieve the patient of the risk of that perforation. That is the position I take.

It is possible to err in the diagnosis of appendicitis, but it is not at all likely that one will err in the treatment by operating in the early stage. That is the safest time and the best time to operate, so far as the patient is concerned.

Another matter of importance is the interpretation of the significance of pus in the peritoneal cavity. What do you mean when you say that there is a pint of pus in the free peritoneal cavity? That pint of pus can be either *defensive* and *protective*, or offensive and destructive. What do we mean by that? When there is an infection of the appendix without a perforation, immediately a transudate forms in the peritoneal cavity, and that transudate contains a colossal number of lymphocytes and polynuclear leukocytes. That is nature's method of defense against infection. That is defensive and protective pus. A little later comes the perforation of the appendix or the rupture of a gangrenous appendix. The débris is poured into that defensive pus. It offers a defense as long as it can, but finally all that pus becomes virulently infected and then it is a destructive or offensive pus. A very short time ago there appeared an article in which the author reported 400 cases of perforative appendicitis with suppuration into the free peritoneal cavity with only a few deaths—all occurring within one year. It is difficult to believe that any man could have 400 cases of that kind in a year. We have had about 92 or 93 cases in seven years —where there was a direct passage of infective products from the appendix into the free peritoneal cavity. He called all his pus cases general suppurative peritonitis. That was wrong, because in the great bulk of these cases the pus is defensive and protective pus, and only occasionally is it destructive when perforation has taken place as already stated.

It was the recognition of the significance of this pus that led us to change the former management of general suppurative peritonitis, namely, washing out the abdominal cavity, sponging off the flakes of lymph from the intestine, handling the intestine, doing a nice, clean scraping, washing, and rubbing job, and sending the patient back to die in a very short time. That is what happened in 1896 when, at the meeting of the American Gynecological Society in Chicago, I made the statement that all these patients were going to continue to die. That was my experience. Then I learned that this pus was often not offensive and that the lymph that was on the bowel was a protective lymph, and that every time I took off that lymph I took off the endothelial cells that are essential to the absorption defense integrity of the bowel, and by removing them I left the lymph-spaces beneath wide open to absorb the infective products. As soon as the patient was back in bed he had an overdose of absorption which killed him, just like an overdose of strychnin would. That led me to look for the point of leakage. That is absolutely essential, as was shown in my paper on duodenal perforations. An author reported 19 cases of perforation of the duodenum, all of them drained without closing the perforation. Eighteen of the patients died shortly after the operation, and the nineteenth died some six months later from a secondary abscess. We all know now that perforations of the duodenum are the most innocent type of perforation in the abdomen. If you close up the perforation and drain, the patient will get well. Sixteen or 18 out of 20 patients recover from the operation if it is done at all timely just by closing the hole. The first essential after you open the abdomen is to find the hole and close it. The next essential is to put a drainage-tube into the most dependent portion of the abdomen, so far as that particular infection is concerned; for instance, if the perforation is in the duodenum, put the tube under the liver into the duodenal and hepatic fossæ. Have the patient sitting up. Do not sponge, do not wash out the abdomen, just put in the tube to relieve the tension on the products of infection, just the same as you would if you had a phlegmon of the thigh. In that what do you do? Incise it and put in a tube. You don't scrape it, because if you do you abraid the surfaces, increase the absorption, and put the patient in a fatal condition. He is already carrying all the load he can possibly carry in the way of absorption. The next thing is to overcome the intoxication that is still present. You do that, if the patient is cyanotic and is severely intoxicated, by an intravenous infusion of normal salt solution, or half of the normal strength, while the patient is still on the table, to dilute the toxic products and then start the saline proctoclysis. Keep that up until you overcome the intoxication. The patient may have a suppuration around the tube for five or six weeks; he may have secondary abscesses form either in the liver or in the hepatic fossa; but you have tided him over until such a time as you can safely open the abscess. The abscess may rupture into the pleura, but when it is encapsulated, you can open the pleura without having his life greatly endangered. You have tided him over until he has established an immunity to the inflammatory condition, as indicated by the high leukocytosis. You have brought him out to a place of safety from a place of danger.

When the first five or six patients recovered after this method of treatment, as they did, we thought it was an accident; then we had 12 or 15 without a death and we thought it was a coincidence. Now we know that they will get well because the treatment is scientifically correct and the result has become a habit. These results will continue to be good if you bear in mind all these things that are necessary to do and do them timely, so that to have a death from general suppurative peritonitis of the perforative variety would be one of the things that would lead us to ask ourselves "why?" when formerly it was a recovery that surprised all of us and led to questions. Since 1896 I have had three deaths. One of them was due to a double pneumonia, another to a thrombophlebitis of the portal vein, and the third patient died from a cardiac lesion. Not one of them died from the suppurative peritonitis.

That did not mean elective cases. That included *all* the cases that come to us for treatment. They are all hospital cases, not private cases. Some one has hinted that we have no severe cases. There is no truth in that, but if it were my abdomen, I would think the condition was severe enough if I had to undergo that method of treatment. All the men who refer cases of appendicitis to us are very generous They bring in the cases early now; in other words, they have come to respect the symptoms and they do not wait to see if perforation is going to occur. When they are perforated cases, and sometimes in appendicitis rupture may occur early,—in one case six hours after the onset of the disease,—they bring them in early, so that we are fortunate in that particular.

The details of the treatment of perforative peritonitis, considered in the order in which appear the pathologic processes that cause the mortality, are as follows:

Septic intoxication is the primary and most frequent cause of death in perforative peritonitis. The prevention of absorption is, therefore, of greatest importance. There is a preoperative period in which much may be done to retard absorption while preparing for surgical intervention.

The preoperative and postoperative importance of the Fowler position is underestimated. The patient should be placed in the Fowler position at his home the moment the diagnosis is made, and kept so until convalescence is well advanced. He should be taken to the hospital in this position to prevent the spreading of the infection to the upper abdomen during transportation, carried so to the operating-room, and operated upon with the shoulders at least well elevated, if, as may be the case, the abdomen cannot be approached in the sitting position. This procedure is based on the physiology of peritoneal absorption, normal and pathologic, and further discussion is not necessary.

It is scarcely necessary to dwell on the importance of peristaltic rest by withholding food (emphasized by Ochsner), or the application of cold to the abdomen for lessening the distribution of the infective material. The use of opiates for this purpose is to be condemned, as the relief from pain experienced by the patient, the apparent improvement in the general condition, and the mild cardiac stimulation produced give a *false sense of security leading to dangerous delays.*

Gastric lavage may be resorted to with advantage immediately preceding the operation if the case is twenty-four hours advanced —the operation to be performed at the earliest possible moment. In intensely poisoned and cyanotic cases a preoperative intravenous injection of two to four pints of normal salt solution materially improves the condition. The line of incision is to be made over the seat of the perforation when its location has been determined; otherwise it is made at the right of the median line, as the perforations are much more common in the right than in the left half of the abdomen, and the incision can be enlarged upward or downward *ad libitum* without dividing muscle-fibers transversely. The manner of going through the abdominal wall is of little moment, and depends upon the individual operator's custom. For appendiceal cases we make the incision through the aponeurosis of the right rectus muscle to the inner side of the linea semilunaris, open the peritoneum quickly, and locate the point of leakage.

If the duodenum or stomach be perforated, enlarge the opening immediately, as it is necessary for free work and avoids traction on the viscera in these cases. If the appendix is suspected, it can be best brought into the field by passing the finger of the right hand outward beneath the abdominal wall to the base of the fossa, then turning inward and upward over the iliac vessels, hooking up the meso-ileum and bringing it into the field. The ileum can be readily followed to the appendix, which saves much time and manipulation.

Closure of the Leak.—If the leak is in the appendix, for the sake of speed clamp it at its base, ligate it in the crease made by the clamp, and amputate; *burial of the stump usually entails too much time and manipulation to warrant its execution.* Return the cecum to the abdomen. If the leak be an intestinal or gastric ulcer, close by a double or triple row of Lembert sutures. In gastric cases, with considerable inflammatory induration about the ulcer, this may be difficult or impossible; in such cases close the opening by stitching a coil of the duodenum or colon over it. *Never permit the opening of a perforation to remain patent*, depending upon drainage for relief. Do not resect gastric ulcers in the presence of perforation. If the pylorus is involved in the induration and obstruction is likely to follow, perform a posterior gastrojejunostomy with the button; in these cases sew the duodenum securely over the perforation, peritoneal side in.

The mortality from simple drainage without closure of duodenal, gastric, and intestinal perforations is such as to brand that line of treatment as inefficient and unjustifiable.

Washing removes the pus, with its myriads of leukocytes, sent there for the sole purpose of protection; if it be soon after the peritonitis has set in, the pus may contain only the harmless Staphylococcus albus. If it be late, washing will endanger the patient, in that it tends to carry virulent colon bacilli to new, unaffected parts of the peritoneum.

Irrigation of the whole cavity prolongs the operation, necessitates more or less evisceration, traumatizes the peritoneum, spreads the infection everywhere, and especially does it carry it to the diaphragmatic zone.

Dudgeon and Sargent believe that streptococcus peritonitis is a clear indication for flushing, and "the most thorough possible." This is advocated because the spreading of such a peritonitis is extremely rapid, and the phagocytes are but little able to cope with this form of infection and may be washed away without robbing the patients of what, in other infections, constitutes the chief defense. Our views on this matter have already been given: *Disturb the protective peritoneal endothelial cells early. Streptococci travel through the peritoneal and into the subperitoneal lymph-spaces freely and cannot be washed out.*

In virulent infections, gastric perforations because of foreign bodies, and intraperitoneal hemorrhage, many writers advise thorough washing with a view to removing the foreign material, gastric or intestinal contents, and blood. This might have advantages if it could be effectually accomplished.

The following case is an illustration of the streptococcus infection: Young female admitted to medical department of Mercy Hospital Sunday evening; had complained of intense pain in abdomen since Saturday morning; leukocyte count, 22,000. Monday morning at 9 o'clock it was 34,000; at 12, when she was transferred to the surgical department, it was 80,000; all these estimates were made by the same microscopist. The abdomen was greatly distended, and a diagnosis of perforative peritonitis was made. On opening the peritoneum it was found intensely congested and edematous, and contained a moderate quantity of free pus which analysis showed was pure streptococcus. The appendix was found to be the atrium of infection, but had not perforated; it was free from adhesions, and was removed as above described; a large drain was inserted down to the pelvis; patient was placed in the Fowler position, and 20 c.c. of streptolytic serum were administered at a single dose, and repeated every twenty-four hours for four days; a continuous proctoclysis was instituted, and 18 to 25 pints of salt solution were absorbed each twenty-four hours. It was a pure streptococcus infection, and we believe came to the appendix from a streptococcus infection which the patient had had in her index-finger two weeks before. Recovery was uneventful. *The relief of pus tension is the first* *surgical step toward retarding absorption in all acute infections.* The maintenance of low pressure in every pus-pocket is a *sine quâ non* to continued freedom from absorption. Reduction of tension *must be initial* and the *absence of pressure continuous.* These purposes are accomplished by efficient drainage.

Drainage.—Fenestrated or split rubber tubes inserted to the stump of the appendix or the site of the ulcer, and into the vesicorectal or Douglas' pouch, and any other pus-pockets that may exist, is the only form of drainage permissible. Glass tubes cause strangulation, pressure necrosis, and kinks that have been a frequent cause of death. Gauze packings, drains, loose or cigarette, cease to be drains in a very few hours and are then a distinct menace. They are to be used only to wall off intestinal contents where necrosis has occurred or is imminent, *and then only in conjunction with tubes.*

As already stated, some operators attribute an especial element of success to their *non-drainage.*

In every position in the body where a virulent pus-cavity has been opened and there has resulted a lowered temperature or a disappearance of fever, the toxic manifestations again arise when the pus becomes retained under pressure.

These principles are axiomatic in the management of acute infections in other parts of the body, and the peritoneum should be no exception. True, the peritoneum has great resistance against bacterial invasion and can care for a larger quantity of mildly virulent infective products without drainage, but if the tension on these products is restored and they are virulent, death after operation will occur on the same basis as preceding it. Therefore, the continuation of the drainage is vital in the virulent cases and will save many lives. The peritoneum should continue to be treated in these particulars on the same basis as infections in other portions of the body. We do not wash, scrub, knead, or traumatize acute osseous or cellular phlegmonous cavities—why the peritoneum? The advice of Callender, over a half century ago, "do not milk an abscess," was admirable, and is applicable to the handling of the peritoneum.

The abdomen is often closed with pints of pus still in the peritoneal cavity; its low pus tension is maintained by the tube, which is stitched to the lower angle of the wound or brought out through the rectus muscle.

The entire technic should be accomplished in a very few minutes, *i. e.*, get in quickly—get out quicker. Time is vital in these cases. If the anesthesia is prolonged, the manipulation great, the sponging and flushing excessive, the patient leaves the table with a feeble capillary circulation and does not rally. It has taken fifteen years for the surgical profession to overcome the evil tendency of slow and prolonged operations advised and practised by the masters of surgical pathology twenty years ago. The records of every hospital in the period from 1889 to 1904 prove the truth of this statement; they read: "Peritonitis—operation—death from collapse."

The pus discharges from the tube for many days in some cases and only for a short period in others. The tube does not become rapidly circumscribed by agglutinations if the pus is virulent, notwithstanding some of the recent teachings, based on animal experiments.

[Dr. Golden performed the operation in the usual manner. The patient made a good recovery.—Ed.]

OSTEITIS FIBROSA CYSTICUS OF THE UPPER END OF THE FEMUR, NOT INVOLVING THE HEAD AND NECK—TRANSPLANT PLACED IN CAVITY

HISTORY

Patient, a male, aged twenty-seven, enters hospital on account of deformity of right thigh. When patient was nine years old (1895) he fell while running and landed on both knees, striking harder on the right than on the left. He could not get up on account of severe pain extending from the right hip to the knee. He was confined to bed for two weeks after the injury, and had sharp shooting pains in the right thigh to knee much of the time. After two weeks he was up and about, but continued to have some pain in the right thigh for the next two months. He did not have either a chill or fever. When he was fourteen years old (1900) he tried to jump, slipped and fell, one leg extending forward and the other backward. He immediately had severe pain in the right thigh, just below the hip. He was unable to rise, and was carried home. For the next two weeks he had acute pain, shooting in character, extending down the right thigh to the knee. The slightest movement of the leg increased the severity of the pain. It gradually became less severe, but he had a dull pain in that thigh for the next three months. At the end of three months he was able to walk and after six months he was free from pain. When the pain ceased, he noticed a change in his gait. The right leg seemed to be shorter than the left, and there was a slight bowing outward of the leg at the junction of the middle and the upper third of the thigh, so that he walked on the ball of the foot. We infer from this history and the skiagrams now obtainable that in the accident he sustained a green-stick fracture and a possible focalization of a low type of infection, which we believe is always the cause of an osteitis fibrosa cysticus. From that time on the bowing has gradually increased in degree and after walking some little distance he has pain in the thigh. He wears an elevated shoe, which has to be increased in height from year to year on account of the shortening of the leg caused by the bowing outward of the thigh. He has never used either crutches or a cane. His right leg is 3½ inches shorter than his left. He has had measles, scarlet fever, whooping-cough, and pneumonia, and a Neisserian infection ten years ago, with three acute exacerbations. No history of lues. Family history negative.

COMMENTS AND OPERATION

Dr. Murphy (Operation May 24, 1913): This is a beautiful case from a pathologic standpoint. When did he have the second injury?

Intern: He sustained the first injury when he was nine years old, and the second when he was fourteen.

Dr. Murphy: How old is he now?

Intern: Twenty-seven.

Dr. Murphy: Thirteen years have elapsed since his last accident. How many years is it since his leg began to shorten?

Intern: Six months after the last accident.

Dr. Murphy: Six months after the last accident he first noticed a shortening of the limb. Shortly afterward he noticed that the upper portion of his thigh was bowing outward, and he had pain if he walked some distance. He wears an elevated shoe which has had to be increased in height from year to year until now he has an elevation of 3½ inches.

The question arises, what is the matter with him? What is the lesion? What was the original lesion? When you look at the skiagram you would say that the lesion in the trochanteric region of the femur looks like a sarcoma. It does, but it is not a sarcoma. After you have become accustomed to interpreting skiagrams you would conclude at once that it is not a sarcoma. You can make a diagnosis in this class of cases as well with the Röntgen-ray as you can with the microscope. Remember that the microscope is merely a more acute eye than the human eye,

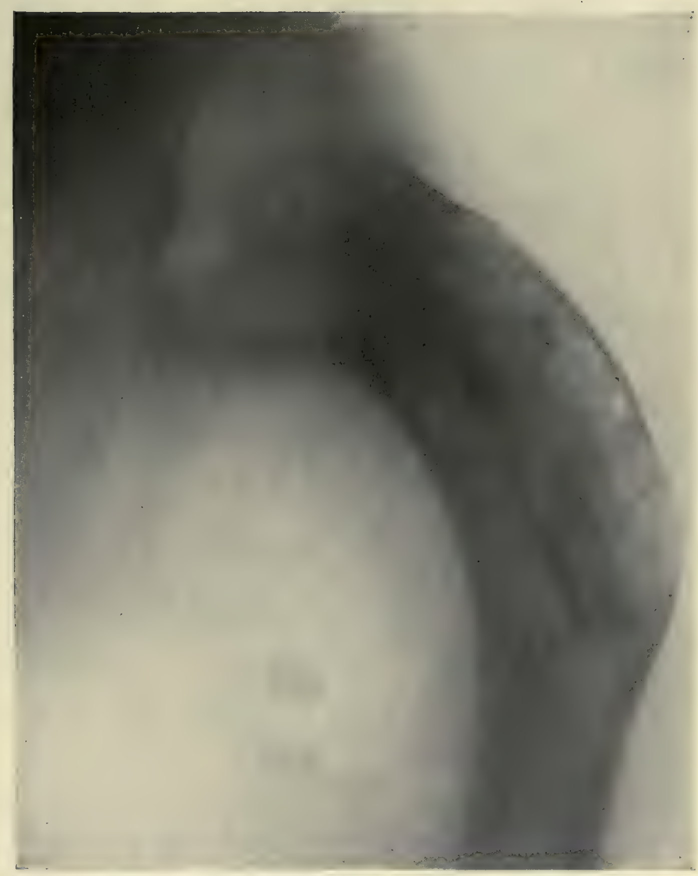

Fig. 194.—Osteitis fibrosa cysticus. Skiagram taken before operation. The tumor extends from the trochanter down on the shaft of the femur for a distance of about six inches. There is considerable outward bowing of the bone. The head and neck are not involved. The structure of the tumor is well shown in this picture.

and to arrive at a diagnosis you must be able to read what you see. What is there about that picture that aids one in making a diagnosis? He has a clinical history of sarcoma extending over rather a long period of time, but the skiagram makes the differential diagnosis and makes it absolutely.

Look at the skiagram. You will note that the femur is enlarged from the middle of the great trochanter downward for a distance of about 8 inches. It is a fusiform enlargement with a considerable bowing outward of the bone at that point. It appears to be hollowed out. There are a number of pockets separated by areas of compact tissue. These pockets are clean cut, differing entirely from the pockets in a sarcoma. The lesion does not extend beyond the periosteum. There is no infiltration of the surrounding soft tissues. The periosteum is slightly thickened but otherwise normal in appearance. It looks like a rapidly growing lipoma. What is it? It is an osteitis fibrosa cysticus. It is a classic picture of that lesion. We have had three other cases just like this—two of the upper end of the humerus and one of the tibia. By comparing the pictures which I show you you can see how much they look alike. In the humerus cases there is an enlargement of the articular surface which is not present in the case before us today because the lesion does not extend into the neck of the bone. It is limited to the region of the trochanter. In the humerus case it extended right to the epiphyseal line. One would imagine that the epiphysis was involved, but it was not. The lesion extended to the metaphysis, but did not invade it.

The history of this case of osteitis fibrosa cysticus was as follows: Miss M. C., aged ten, admitted to Mercy Hospital October 11, 1910. The skiagrams in this case show the nature of the disease. The diagnosis was osteitis fibrosa cysticus. The bone first undergoes a fibrous degeneration; then there is liquefaction of the fibrous deposits, producing small cystic cavities in small areas, while a fibrous mass is present in others. This patient had been subjected to mercurial treatment which yielded negative results (Figs. 195, 196, 197, and 198).

Treatment.—Operation October 14, 1910. An incision was made along the anterior border of the bicipital groove, extending down below the attachment of the deltoid. The muscular attachments were separated and retained in position for recognition. The bone was then freed from all its surrounding tissue, leaving the periosteum attached to the excised bone. Six and a half inches were excised, including the head and epiphyseal line. A 7½-inch transplant was prepared from the crest of the tibia; the periosteum was retained. This transplant measured ⅞ by ½ by ½ inch. The medulla of the remaining portion of the shaft of the humerus was reamed out and the transplant inserted for ¾ inch. A nail was then passed through a drill-hole transversely to prevent it from embedding itself deeper. The upper end of the implant was inserted into the glenoid cavity, and the capsule accurately sutured around it. The muscle-ends were then attached by an encircling suture around the implant in about their normal anatomic position (Fig. 199). The wound was closed by a deep row of catgut sutures and superficial horsehair sutures. The arm was dressed in abduction at about a right angle to the body. A Buck's extension was applied to the forearm, and a 10-pound weight attached with a line running from a pulley. This was to prevent muscular contraction during the period of regeneration, which would probably drive the upper end of the bone through the capsule. It was kept on for five weeks.

Result.—There was complete primary union, and at the end of the five weeks the humerus appeared on palpation to be as large as the normal humerus. The patient had good voluntary motion. The position to which she could extend her arm when she left the hospital (November 16, 1910, five weeks after operation) showed how much abduction was produced by the deltoid and supraspinatus muscles. A subsequent skiagram shows how completely this bone regenerated, not only in the part outside of the capsule, but also how it has filled in within the capsule. It also shows the periosteum remaining intact, and not becoming ossified even after this date, so that it would appear that the periosteum on the transplant is a detriment rather than an advantage. At least it continues to be a limitator of osteogenesis at that zone. Serial skiagrams of this case were taken every few weeks showing the condition, so that a careful study could

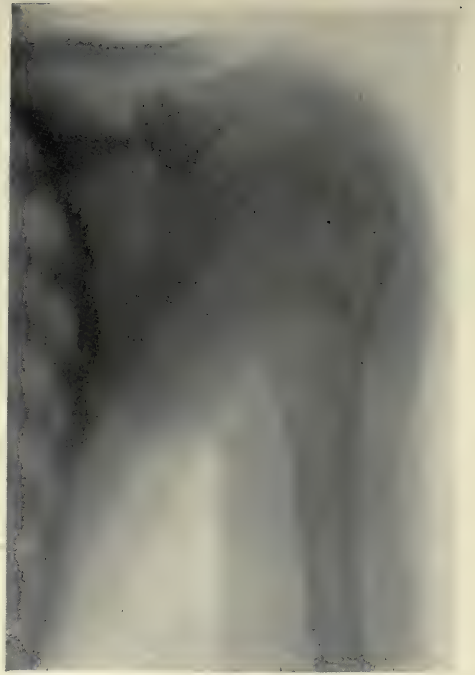

Fig. 195.—Skiagram made about nineteen months before operation, showing the disease involving about three inches of the upper end of the humerus.

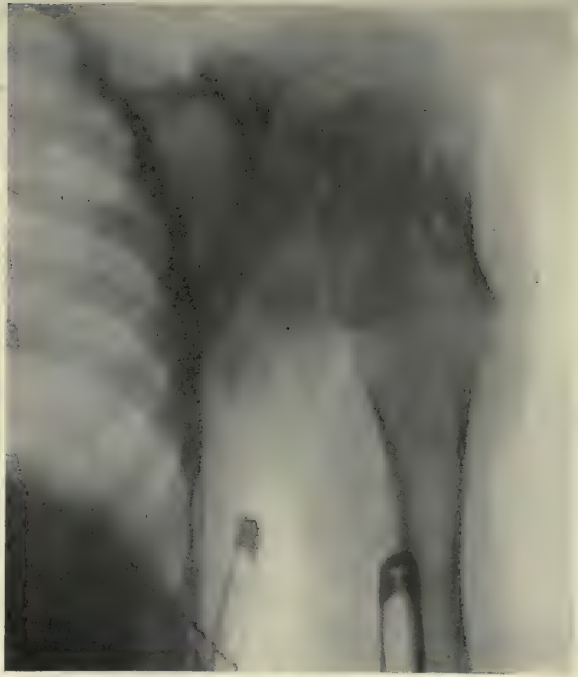

Fig. 196.—Skiagram made six months before operation, showing the progress the disease made in the preceding thirteen months. It now involves about four inches of the upper end of the humerus. The structure of the tumor is well shown.

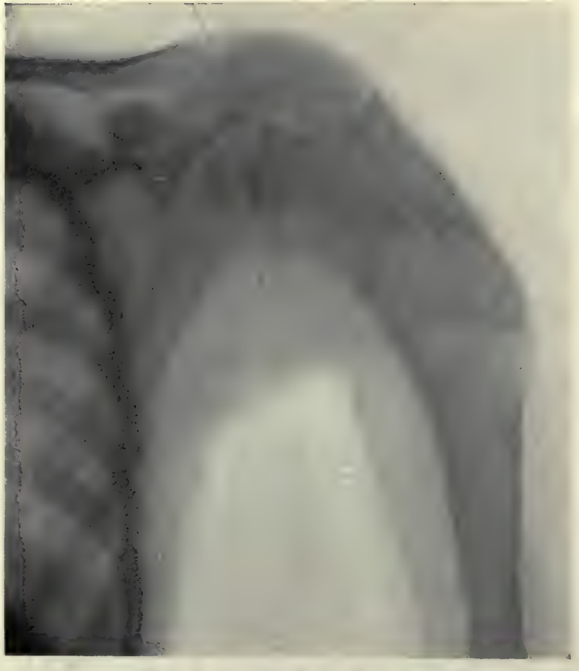

Fig. 197.—Skiagram made three months before operation. The humerus has now become bowed outward, and the upper end of the humerus is considerably enlarged.

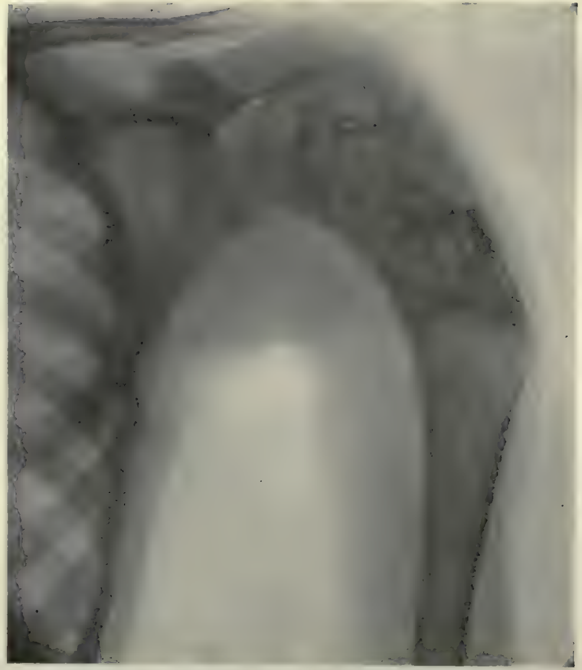

Fig. 198.—Skiagram made two months before operation. By comparing these four skiagrams the progress of the disease is well shown. The articular surface of the head of the humerus does not appear to be involved at any time. The epiphyseal line is well shown, and the change in its direction occurring with the growth of the tumor is marked.

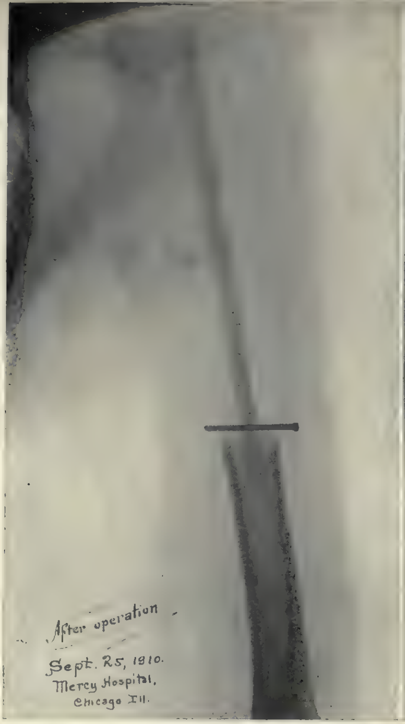

Fig. 199.—Skiagram made immediately after the operation. The lower end of the transplant is impacted in the medullary canal, and the remaining portion of the shaft of the humerus is fixed with a small wire nail. The upper end of the transplant is in the glenoid cavity, the capsule of the joint having been sutured around it.

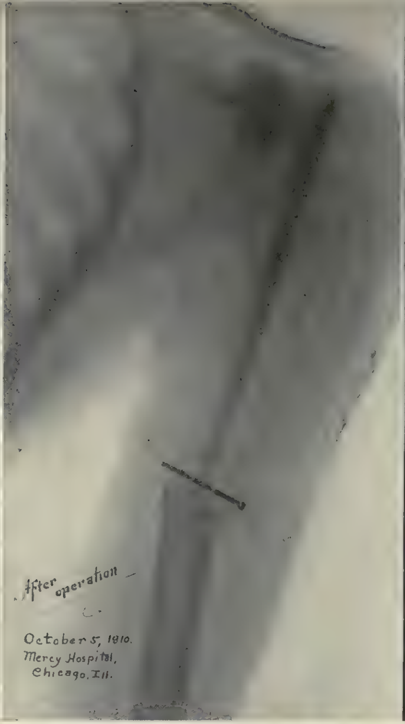

Fig. 200.—Skiagram made eleven days after the operation. The periosteum surrounding the transplant, which was not removed with the bone at the time of the operation, is shown very well in this picture. The muscle traction has produced a slight angulation where the transplant was inserted into the shaft of the humerus.

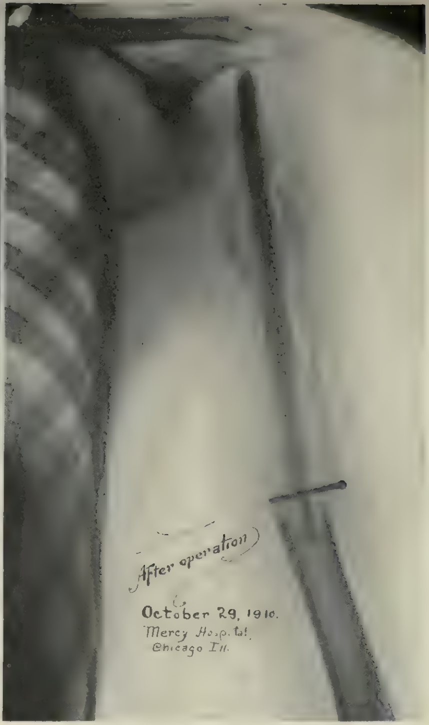

Fig. 201.—Skiagram made about five weeks after operation. The arm is a little straighter than in the preceding skiagram. Bone regeneration is progressing well, and the periosteum is still plainly to be seen.

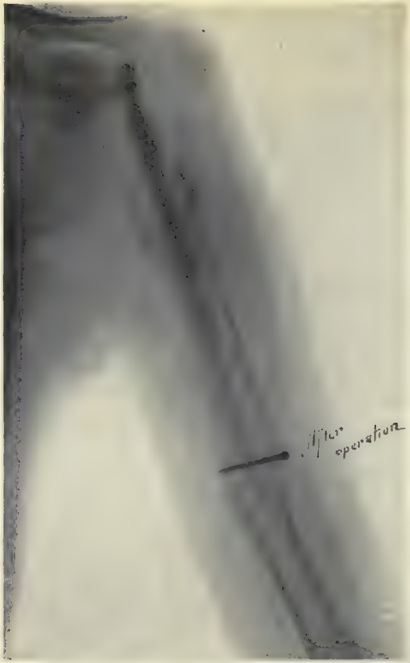

Fig. 202.—Skiagram made about ten weeks after operation. There is a considerable formation of new bone, extending upward from the remains of the shaft of the humerus. In that the new bone appears to be of sufficient size to replace the old shaft. The upper end of the bone is thicker, evidently for the purpose of reforming the tuberosity of the humerus. This is the result of muscle action. The lighter shadow,—almost a white line,—which shows plainly to the right of the transplant, represents the periosteum of old bone.

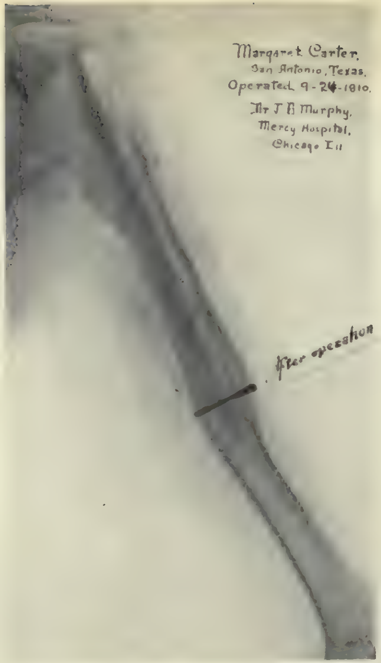

Fig. 203.—Skiagram taken sixteen weeks after operation. Bone regeneration is progressing rapidly.

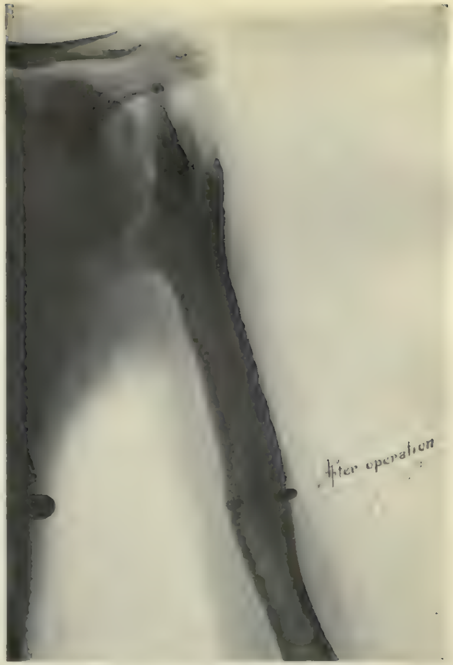

Fig. 204.—Skiagram taken about seven and one-half months after operation. The upper end of the humerus has been regenerated to a considerable extent, including the tuberosity and the articular surface. The white line a little to the right of the central axis of the new part of the shaft represents the periosteum which was left on the transplant. The new bone is slightly thicker than the old bone.

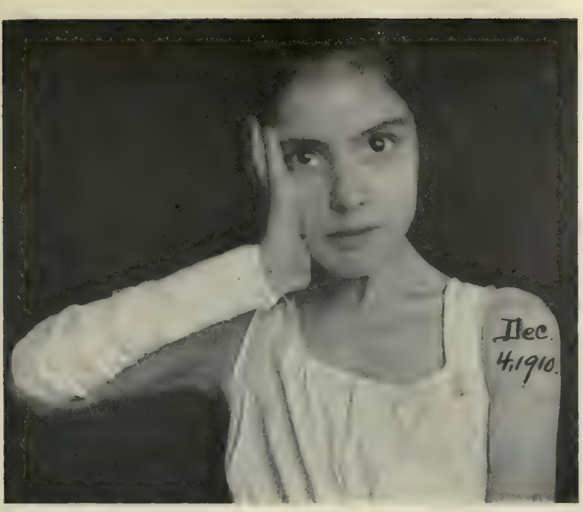

Fig. 205.

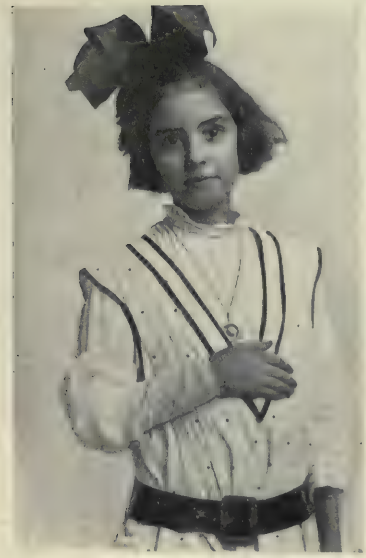

Fig. 206.

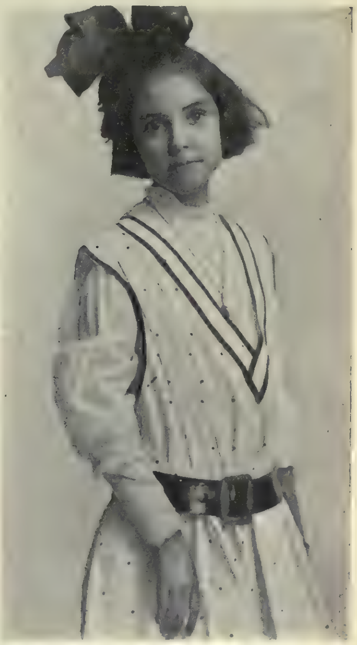

Fig. 207.

Figs. 205, 206, and 207.—Photographs of patient taken about ten weeks after the operation. The patient is able to bring the arm up to a right angle with the body, placing hand flat on face, again placing forearm on chest, showing that adduction as well as abduction is normal at this time. The arm also hung by the side normally.

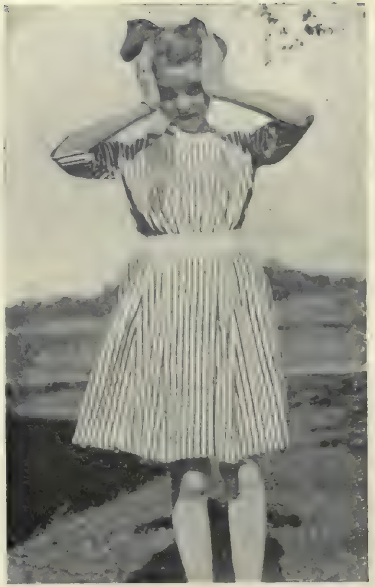

Fig. 208.

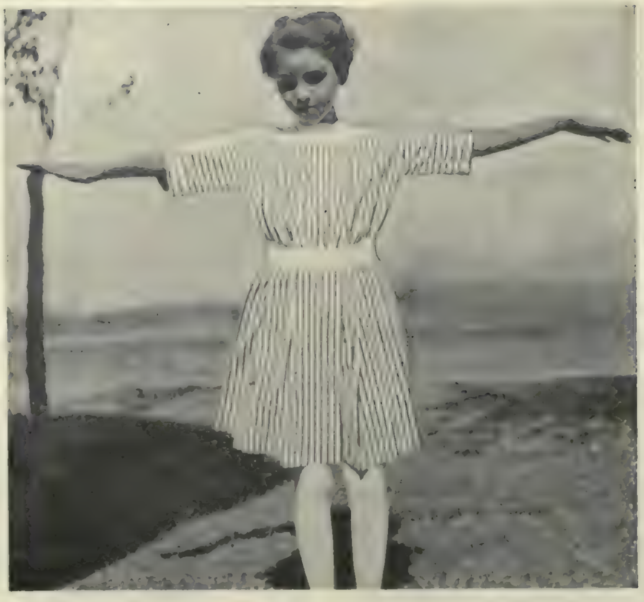

Fig. 209.

Figs. 208 and 209.—Photographs made one year after operation. Flexion and extension are perfect. There is no difference between the normal arm and the reconstructed arm.

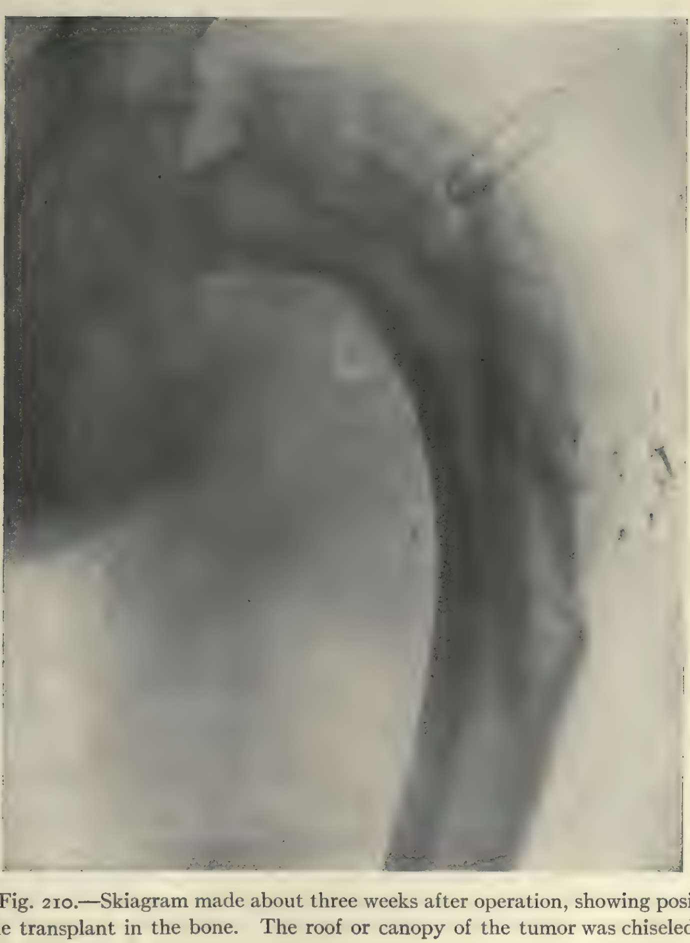

Fig. 210.—Skiagram made about three weeks after operation, showing position of the transplant in the bone. The roof or canopy of the tumor was chiseled off, as is seen in the picture.

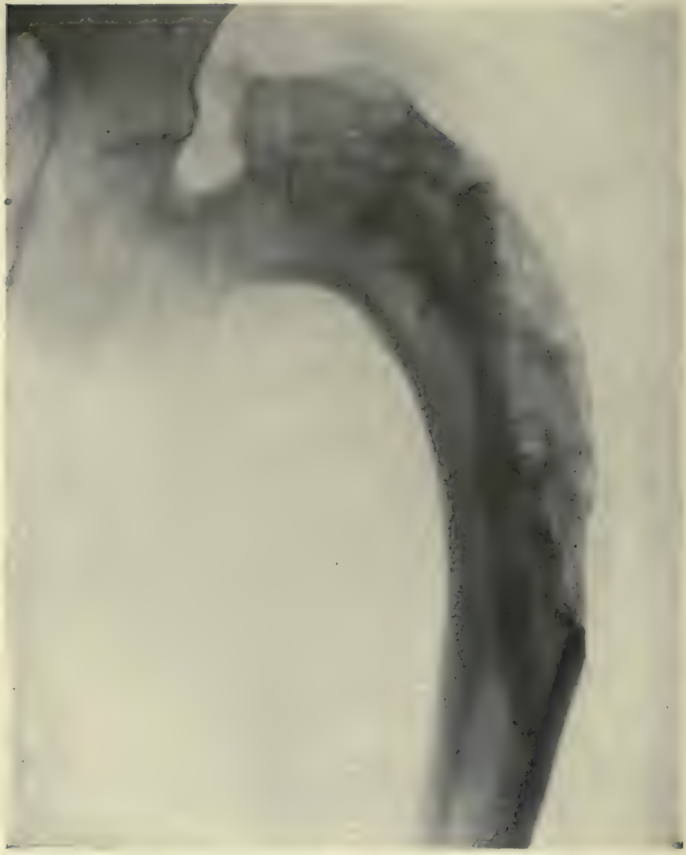

Fig. 211.—Skiagram made about seven weeks after operation. New bone is forming around the transplant, and the femur appears to be straightening out, that is, there is less bowing. It is probably due to the extension which was kept on the leg all the time.

be made of the progress of bone development. Extension from the living periosteum below was the most important; next from the medulla. This rapidly shot upward around the transplant to the capsule of the joint and later into the capsule (Figs. 200, 201, 202, 203, and 204).

The progress was then more slow. She can now abduct her arm to a right angle in extension, and flexion is likewise complete, so that she does not know that any serious operation has been done on her arm, so far as its usefulness is concerned (Figs. 205, 206, 207, 208, and 209).

By studying the skiagrams one can observe the stages of reproduction very well. At the end of five weeks, on palpation, the humerus appeared to be as large as a normal humerus, although the skiagram showed small calcareous deposits only here and there. The development of compact bone proceeded along definite lines—from extensions of the periosteum from the medulla, through the implant, up to the capsular attachment and into the joint, as shown in the last skiagram picture, taken seven months and some days after the operation (Fig. 204).

Here also can be observed the evolution of the tuberosities at the points of muscular fixation. Again, it will be noted how the transplanted periosteum remained as a white line for seven months after the implantation, and that it was an apparent detriment to the production of bone. It was finally involuted by bone formed from the periosteal zone helm at the nail upward around the new-formed callus and outside of the transplanted periosteum.

This shows how completely the humerus can be regenerated and how perfectly function of the extremity can be restored (Figs. 208, 209) in cases in which formerly amputations were made or very defective functional and anatomic results were obtained from excisions, curetments, etc.

A study of skiagrams from dozens of cases of bone transplantations gives one a better idea of the factors of osteogenesis than some of the meager experiments recently reported and used as a basis of estimation. The German literature of the last two decads abounds with carefully prepared and logical articles, with innumerable microscopic plates, supporting these skiagraphic observations.

Dr. Golden operated on two of these cases. (For report of one of these cases see Clinics, October, 1912, p. 741.) In all three we resected the mass and transplanted a piece of bone with periosteum removed from the crest of the patient's tibia. The result was entirely successful. By comparing the skiagrams of these cases one can see at once that these cases are all of the same character; that this is not a sarcoma, but a chronic osteitis with cyst or cavity formation, known in the literature as osteitis fibrosa cysticus. First, the bone undergoes an absorption of the lacunæ and canaliculi, a fibrous hyperplasia, and then a cystic degeneration, forming these pockets, differing entirely from the medullary tumors or cysts and from sarcoma. It is not a malignant lesion at all. It can be removed and not recur, even when the periosteum is preserved.

What is the cause of the trouble? I believe that it is an infection, although I cannot give you any proof. I base that belief on the clinical history and the pathology of these cases. An Italian clinician in the General Hospital in Rome has demonstrated an infection in the human which could be transmitted to the rat, and which produced some kind of an osteitis with absorption of the new formed osseous tissue, usually beginning in the metaphysis. It is a metaphyseal lesion more frequently than a diaphyseal lesion. It is never an epiphyseal lesion. It practically always occurs in the shafts of bone, corresponding in that regard with acute infective osteomyelitis. It goes on and on, increasing in size as it progresses along the bone. The progress of the disease was beautifully illustrated in the case cited above, showing the rapidity of growth and production of the deformity in a little over a year and a half. It is striking. If you expose the tumor, break down its canopy, curet, and clean out radically all the soft granular tissue to be found in these pockets, it does not continue its growth. In our earlier cases we feared malignancy and resected the tumor with the periosteum. Now we curet thoroughly, just as we do in cases of bone-cysts, and put a piece of bone removed from the crest of the tibia into this cavity. Then we put an extension on the limb to prevent muscular contraction during the early stages of healing, which might break the thin shell of bone and cause a fracture of the bone. The fragment of bone in the cavity acts as a transplant and favors the production of bone, until sufficient is produced to fill in the cavity. This patient asked me, "Will you fracture and endeavor to straighten the limb?" Not at all. Why? Because if I fracture that bone and endeavor to straighten it, I would have a great deal of trouble to overcome the shortening. The bone may break accidentally while I am operating, because this shell of bone is very thin. I am going to clean out these cavities thoroughly, break down the partitions between these cysts, and transplant a piece of the crest of the tibia into the cavity. That will favor osteogenesis. We will not remove the periosteum nor the shell of compact bone underneath it. After that portion of the bone has become ossified, say at the end of a year and a half, then I will take up the proposition of correcting the deformity. I will not take out the end of this bone because I know that this tumor is not malignant, and we have enough of the bony framework left to prevent a fracture. The period of time necessary for the reproduction of bone in this case will not be very great. In three or three and one-half months he will have a fairly compact bony mass filling in this cavity.

There is another type of tumor of bone in which one cannot preserve the periosteum, because the tumor is a malignant one. We had a case involving the head and neck of the femur and the upper portion of the trochanter. It was a traumatic chondrosarcoma. We excised the upper 5½ inches of the femur, including the head, neck, and trochanters, with the periosteum, and transplanted a fragment of bone taken from the crest of his tibia. The lower end of the transplant was driven into the medullary canal of the femoral shaft; the upper end rested in the acetabulum. When the transplant was in position and the extension put on the leg to elongate and stretch the muscle, we had to replace the muscles attached to the trochanter. This we endeavored to do by fixing them to the transplant with a nail driven through it. In carrying out this procedure the nail split the transplant. I feared that the upper end of the transplant would die *because it was not in contact with the osteogenetic living bone* and, therefore, it apparently violated a law I had laid down concerning the regeneration of bone. The upper end of the transplant, remember, was in the acetabulum; the lower end of the upper fragment now was merely contacting with the lower fragment of the transplant, but fixed firmly to it with catgut. That operation was done October 8, 1911, and in December, 1912, the skiagram showed the bone regeneration passing from one transplant fragment to the other. The fracture of the transplant did not prevent bone regeneration. What, at the time, I believed would prove a detriment was a blessing, because at the seat of the transplant fracture he has developed a fairly good trochanter. That forces me to make an addition to the law I laid down with regard to bone regeneration, namely, that bone may regenerate if the transplant is put in contact with a graft that has been revivified, and that it will finally take on the normal appearance of bone. Whether it is a regeneration of the same bone-cells or not does not matter to us as surgeons. The important point is, as shown by our results, that if you will place a separated transplant fragment in contact with a living transplant, it becomes revivified and ossification goes on, just as it does ordinarily. Remember, principles are convincing. One case can establish a principle.

We also have had a case, in a girl sixteen years of age, a giant-cell sarcoma of the upper end of the tibia, but not involving the articular surface for its entire transverse diameter, but it did involve it fully at the internal tuberosity. All the diseased tissue was removed, but the epiphyseal line and the portion of the epiphysis close to the fibula were retained. I took out the periosteum, of course, because this was a malignant disease, and implanted a fragment of bone, 5 inches long, from the crest of the opposite tibia. The skiagrams made eighteen months after the operation show a very good restoration of the bone, with no evidence of return of the neoplasm. Let us apply that principle of treatment during the first six months of a patient's illness as soon as we make a diagnosis of a malignant disease. The patient will then consent to a removal of the upper end of the tibia and transplantation when she would not consent to an amputation of the leg. We are not certain that the case I have just cited is going to be cured, because a sufficient time has not elapsed since the operation to obviate entirely the recurrence of the disease either in loco or elsewhere. If you get these cases in the early stage of the disease, before metastases would probably have occurred, you can say without hesitancy that the patient has been saved not only the limb, but life.

In the early stage of a malignant bone lesion we can always leave the epiphysis, because the disease is slow to transgress the epiphyseal line, which is cartilaginous and forms a line of demarcation for a goodly period of time, since cartilage greatly resists infection and neoplastic invasion. Another very important point in this work is absolute asepsis. This class of work requires a much higher degree of asepsis than is carried out in peritoneal or other surgical work. If your asepsis is not perfect, you are in trouble all the time. The occurrence of infection may be disastrous to your work,—even destroy life,—so that one must exercise the greatest care from an aseptic standpoint.

As to the case before us, if one could attack these lesions early, before the disease had involved so much bone, it would not be hard to treat them. It is not easy to get a transplant to fill so large a cavity. I am certain that I shall not have to take off the trochanter. The trochanteric muscles will not need to be disturbed. We will make the incision on the outer aspect of the thigh, between the groups of flexor and extensor muscles, and directly over the lesion, and by retracting the soft parts, the tumor is plainly in view. I will incise the periosteum over the tumor and lift it off the bone with the periosteotome because we want to save it. You can see how readily that periosteum came off. It appeared to have a very feeble attachment to the bone. Now we are ready to break down the canopy or roof of the tumor with a chisel, so that we can expose the pockets in it and clean them out thoroughly preparatory to inserting the transplant. The arching or bowing outward of the femur makes it difficult to insert a transplant. It will have to be put into the cavity on the slant. One can hardly make a curved transplant. We must avoid too much manipulation or injury of the tissues because every trauma under an anesthetic is equivalent to the same trauma without an anesthetic, so far as this bone work is concerned.

These cavities are lined with granulation tissue, which we will remove very thoroughly with the sharp curet. Then, with the chisel, we will break down a part of the wall of some of these pockets so that we can place the transplant and have it immobilized. There is compact bony tissue here, forming the outer wall of the tumor. There is a large pocket right under the trochanter. You can tell by the sound of the chisel scraping on that bone that it is hard, compact, new-formed bone. That is not the kind of bone present in a sarcoma. It is like the bone produced when an infective osteomyelitis has healed. It is a cicatricial bone.

There are a number of areas here undergoing mucoid degeneration. These areas look like tubercular "brie." The absorption that is present here resembles the type of absorption one sees in a sequestrum that is being separated from the living bone. That means that this is an active and not a passive process. Now we have this cavity well excavated. It is quite large, measuring about 7 inches in length and 2 inches in width in its widest portion. We handle this limb with more care than usual, because we are liable to fracture the femur, which is very thin here—just a mere shell. The transplant which we will place in this cavity must be 7 inches long. We will take it from the crest of the tibia of the other leg, because we must put a Buck's extension on this leg and therefore cannot have a wound over the tibia.

When exposing the tibial crest for the purpose of removing a transplant, never make the incision directly over the crest. Make it one-half inch to the outer side, over the tibialis anticus muscle. Cut the aponeurosis of this muscle, and retract the muscle downward. That gives us a good exposure of the tibial crest. I cut the periosteum with the scalpel on both sides of the crest and nick the bone above and below to mark the length of the transplant. We always retain the periosteum on the transplant. Why? Because by this method we have had 100 per cent. of successes and there is therefore no reason for changing our technic. We do not believe that the periosteum is a necessity—merely an advantage. It serves as a limiting membrane and not as an osteogenetic factor. In one case (Miss M. C., cited above) it was apparently a detriment to osteogenesis, as is shown by the skiagram. The periosteum appears as a white line months after the transplantation. The retractor we use here is one of the few special instruments. It not only keeps the soft tissues out of the field, but serves as a guide for the chisel or saw used to remove the transplant. We have very few special instruments for this work. With the motor buzz-saw I can remove the transplant in about one-third the time required with the chisel. This transplant measures 7 inches in length by ½ x ¼ x ⅜. It is taken right out of the crest of the tibia, exposing the medullary canal. We pry it loose with a large flat chisel, and lift it out of its bed with two sequestrum forceps, as it might fly out of the grasp of one instrument. We keep two forceps on that transplant all the time. It lessens the likelihood of its slipping away. This wound is closed by approximating the cut edges of the muscle aponeurosis with plain catgut and the skin-edges with horsehair. The defect in the tibia fills in rapidly—as we have had occasion to observe in two cases where we had to expose the tibia again subsequently to remove a second transplant because of a fracture of the first transplant months after the original operation, through carelessness on the part of the patient.

Now we will place the transplant in this cavity. It is a snug fit, so that this piece of bone will be absolutely immobilized. Next, I turn in the edge of the vastus externus muscle into the cavity by catching its aponeurosis with the needle away back from the cut edge as I suture the edges of the aponeurosis together. This will not only lessen the size of the cavity, but will fill in the top with muscle instead of taking off the canopy and dropping it into the cavity as one would do in a case of ordinary bone cyst.

In our method of handling these bone infections or bone cysts we first make a crease with the saw or chisel in one side of the canopy. Then we break down the edges of the canopy and drop the bone right into the cavity. By filling the cavity in that way we lessen the period of convalescence in these cases of large bone cavities and hasten osteogenesis. (See August CLINICS.)

We will now suture the fascia lata and fat. We did not disturb the trochanteric bursa with our incision, so that there is practically no danger of its becoming infected. The skin-edges will be approximated with horsehair.

Let the record show that there was a great excavation of the upper end of the femur extending up into the base of the neck; that this had on its outer side a compact bony shell; that there were partitions through it separating it into many compartments or pockets; that these partitions consisted of compact bone; that the wall of the cavity everywhere was compact bony tissue, thus differing from a malignant lesion; that these cavities were thoroughly cureted from one end to the other; that the middle portion of the canopy, about one-half inch wide, was removed so as to give us access to the cavities; that the medulla below was separated from the lower cavity by a partition of compact bony tissue; that a bone fragment 6 inches long was removed from the crest of the tibia of the opposite leg; that it was implanted into the cavity, both ends being fixed under the bony shell first by inserting the lower end into the cavity below and then grasping the transplant with forceps and carrying the upper end under the shell of the trochanter, so that it was held firmly in position at both ends and did not need to be nailed; that when that was completed, an involution was made of the muscles into the cavity down to the transplant; that these were held in the cavity by stitches placed into the aponeurosis, an inch behind the line of division in front, and one-half inch behind the line of division behind, so that we had a fairly good involution and a fairly good filling of the cavity down to the transplant; that the periosteum of the transplant was turned outward. The usual dressing was applied. A Buck's extension with 25-pound weight attached will be put on this leg.

These cases, we believe, are infections with absorption of the bone, but an infection of a low type in which the products have a stimulating effect on the osteoclasts which absorb the bone, or an inhibiting effect on the osteoblasts. The epiphyseal line has a cartilaginous wall which resists invasion and protects the epiphysis. The indications for treatment are clean cut.

From this time on there is just one source of danger, namely, have we admitted a sufficient number of microörganisms to infect the transplant or to infect the clot? A clot in the bone is not a good culture-medium as long as it is a primary one. The danger of infection does not come in the primary clot—it comes when it begins to break down at the end of three or three and one-half weeks.

If that transplant lives,—and we believe it will,—in but a short time there will be a very strong mass of new bone here, making the femur as strong as it ever was. With the strengthening of the bone will come the absorption of the excessive callus, the same as you get an absorption of the ends of an excessive callus following fractures. The final result will be normal, compact, bony tissue. If the shortening is annoying to the patient, we will operate on him again later and endeavor to overcome the shortening.

[This patient remained in bed, with the Buck's extension on his leg, for seven weeks. The wound had healed per primam. The stitches were removed on the seventeenth day, when the first dressing was made. After seven weeks the patient was allowed to be up and around on crutches. He had neither pain nor discomfort in the leg.—Ed.]

CAVERNOUS ANGIOMA OF THE THIGH

HISTORY

Patient, female, single, aged seventeen, enters hospital on account of a mass the size of a walnut on the outer aspect of the left limb, midway between the knee and the hip. She first noticed this mass three years ago, and does not think it has increased any in size. It is very tender; the overlying skin is not adherent to it, but the tumor does not move on the thigh. It is very painful, the pain extending downward on the leg and upward to the hip. She has never had chills or fever. Her tonsils were removed in 1909. Menstruation irregular, three to six weeks; one week's duration; pain in back on first day. No leukorrhea. Family history negative to tuberculosis and carcinoma.

COMMENTS AND OPERATION

Dr. Murphy (Operation May 3, 1913): What is the matter with this patient? The position in which that tumor is located convinces us that this is not a dermoid. It is not a movable tumor. If it were in the rectus muscle, we would say it was a desmoid. It is not adherent to the skin, therefore it must be a muscle or fascia tumor, or it may have sprung from a nerve or from blood- or lymph-vessels. If it is a sarcoma, it originated in the muscle aponeurosis or intermuscular fascia. It might be a metastatic infection in the aponeurosis. That is possible, but not probable, because she has not had any clinical manifestations of infection. It is impossible to make a diagnosis, but the patient has pain from which she must be relieved. Therefore we will remove the tumor first and make a diagnosis afterward. She first noticed this mass three years ago. If it is a sarcoma, it is one of the least malignant types, because muscular sarcomata grow more rapidly. It has been painful, however, all the time. Another point against its being sarcoma is the fact that it is not connected with the bone: it is a part of the soft tissues.

Sarcomata in this region are usually periosteal in origin. It is not a neuroma or neurofibroma and probably not a neurosarcoma. The position of the tumor and the absence of intense pain on pressure rule these out. It might be a pure fibroma, although I do not believe it is. Fibromata of the thigh usually form soft, lobulated masses which may hang in folds. However, the final diagnosis in this case must be made microscopically.

We make a longitudinal incision just to the outer side of the tumor. It is a soft round mass. It looks like a retention cyst, but it does not fluctuate. There is no muscle tissue in it. It seems to be encapsulated. It originated in the aponeurosis between the muscles. I will free that aponeurosis just above and below the tumor, so that I can take it out. We can do that without traumatizing the tumor at all. There is perfectly normal muscle above and below the tumor. There! it is out. I do not know what it is. It looks like a lymphangioma—like one of the cavernous lymphangiomata. We will have some microscopic sections made of it. It does not look like a neuroma, but more like a cavernous angioma which began on the posterior aponeurosis of the quadriceps extensor. On section, that belief is substantiated. It is a cavernous angioma.

Let the record show that the tumor originated on the anterior surface of the posterior aponeurosis of the quadriceps extensor.

We close the wound by approximating the skin-edges with horsehair, sponging with alcohol, dusting with bismuth subiodid, and sealing with collodion gauze. The patient will be kept under observation until after the stitches have been removed.

[Stitches removed on eighth day. Primary healing. Patient left hospital on tenth day.—Ed.]

SARCOMA OF THE THYMUS GLAND

HISTORY

PATIENT, female, aged sixty-nine, enters the hospital on account of a large mass on the anterior chest-wall, in the midline, just below the root of the neck. Twenty years ago she noticed a small hard mass to the left of the median line of the chest on a level with the third rib. The mass increased in size very slowly. Ten years later she noticed a similar mass to the right of the median line. These two masses gradually approached each other and seemed to coalesce. About one year ago she noticed a third mass in the midline above the other two. This mass has been growing rapidly in size, especially in the past three or four months. At present there is a large, pyramid-shaped tumor pointing toward the chin, irregular in shape and consistence, but definite in outline. The base is hard and fixed to the sternum and costal cartilages, with areas of softening above. The large lobe pointing toward the chin is very hemorrhagic and soft. She has never had any constant pain, but has had occasional twinges of sharp pain. The mass does not pulsate.

In the autumn of 1911, from about the latter part of October until Christmas, she had a continuous hematuria, passing large clots of blood as well as bloody urine. A catheterized specimen was not examined. She was in bed six weeks. Says she had no pain over the kidney region at that time or at any time since. She does not think that the chest tumor enlarged any faster immediately after this bleeding.

She has had four children; normal deliveries. Menopause at thirty-five. No flow or uterine hemorrhage since. One son died of tuberculosis five years ago. He was sick four years, and during this time she nursed him. Tuberculin reaction negative. She has lost no weight; for the past six or seven years she has had night-sweats, which would last two or three months and then disappear for a while. She had no night-sweats in the past eight weeks. She has had occasional afternoon temperature elevation for three or four years, but none in the past year and a half.

COMMENTS

DR. MURPHY (Examination May 27, 1913): The trying part of this case is, first, the differential diagnosis; second, after you have made the diagnosis, what can you do for the patient?

The question is, is this a lesion of the sternum or is it a mediastinum lesion which has produced a pressure necrosis of the sternum and is now under the skin? It is not rapid of growth in either case. If it is a mediastinal growth, where does it originate? It was first noticed by the patient twenty years ago. It was then situated to the left of the sternum. Ten years later she noticed a similar mass to the right of the sternum. That gives you an idea as to its origin. It appeared on one side of the sternum first and then on the other, and then a third mass appeared in the middle of the sternum, above and between the other two. When did she first notice the third mass?

INTERN: A year ago.

DR. MURPHY: That mass has grown rapidly? Especially when?

INTERN: In the last three or four months.

DR. MURPHY: Does it pulsate?

INTERN: No.

DR. MURPHY: That is the most important point. It does not pulsate. It is not an aneurysm. With a growth in that position the first thing you think about is an aneurysm. Was there any trauma of the chest at any time in her life?

INTERN: No.

DR. MURPHY: Are you certain about that?

INTERN: Yes.

DR. MURPHY: You heard the history. You see the position in which the tumor is located. The urine examination made yesterday was negative. Late in 1911 she had hematuria and pain over her kidneys. The first proposition is to eliminate the

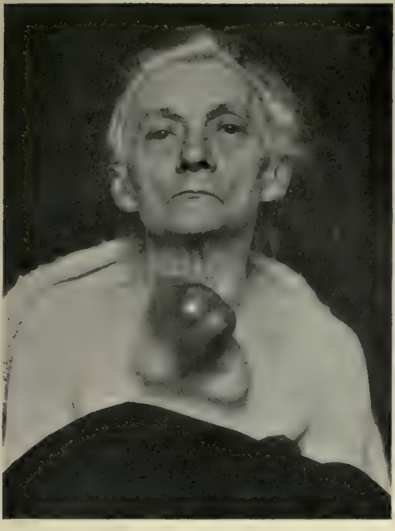

Fig. 212.

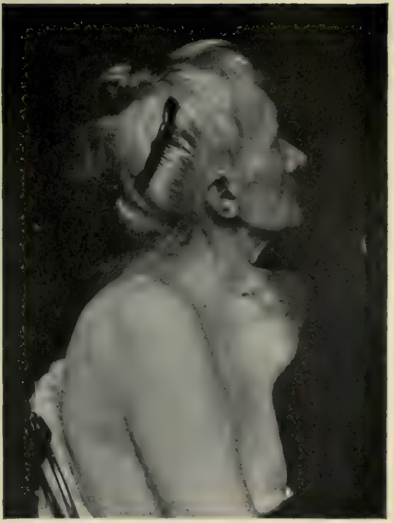

Fig. 213.

Figs. 212 and 213.—Aneurysmal sarcoma of the thymus. Anteroposterior and lateral views, showing the trilobulation and elevation of the tumor on the chest-wall.

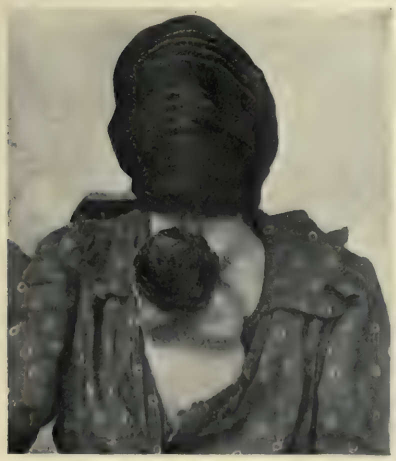

Fig. 214.

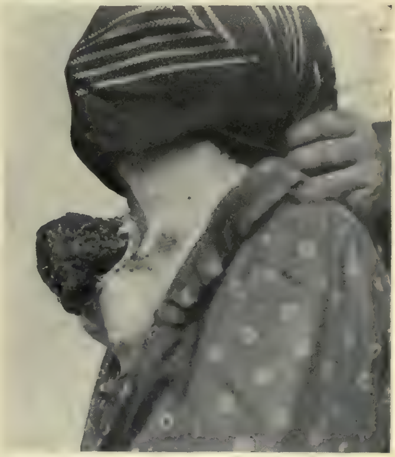

Fig. 215.

Figs. 214 and 215.—Anteroposterior and lateral views, made about five weeks after examination. The skin over the upper lobe of the tumor has become necrotic.

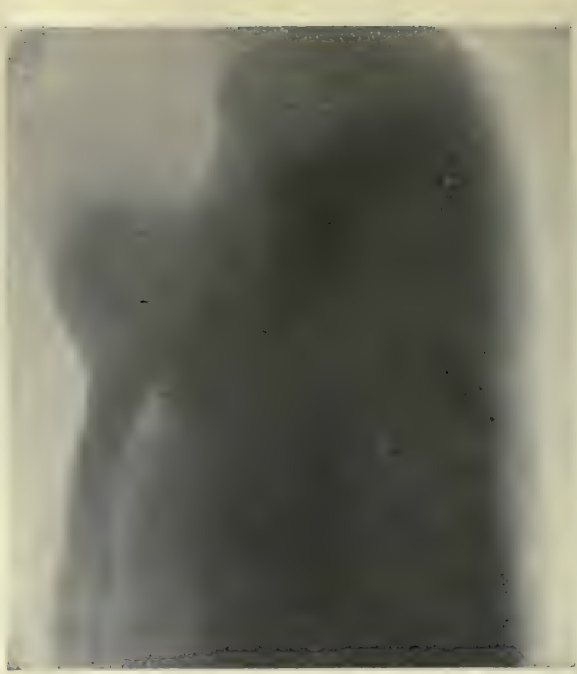

Fig. 215*a*.—Sarcoma of thymus. Lateral view, showing the tumor extending from the thoracic cavity forward and beyond the chest-wall. The shadow is of a uniform density, except in the upper left, hand quadrant, where it is apparently lobulated and gradually becoming dimmer.

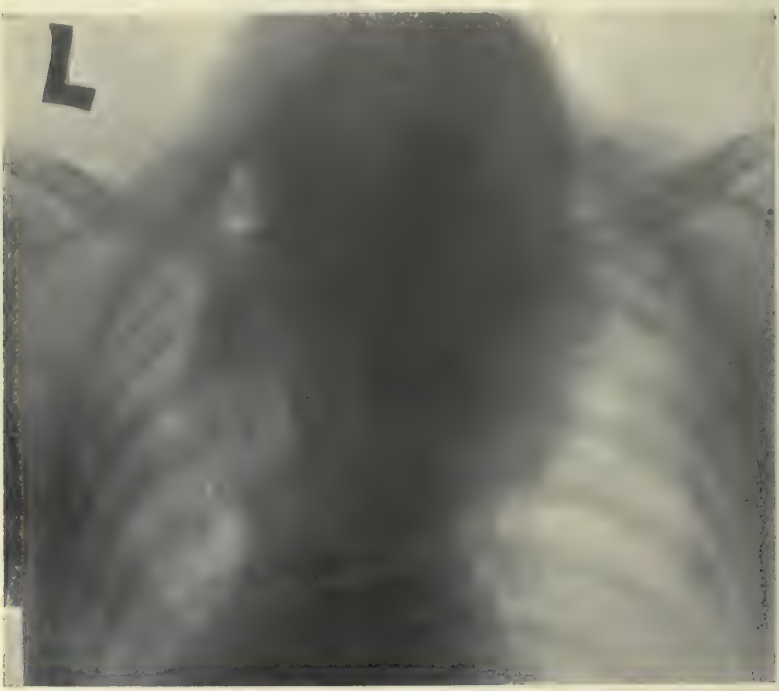

Fig. 215*b*.—Skiagram of the tumor viewed from the anterior surface. Showing extension in the thoracic cavity, with obliteration of the mediastinal space. Note the difference in density between the lateral and superior lobes.

possibility of the kidney being connected with the chest lesion. Is this a metastatic hypernephroma in the sternum? The lesion when it began came through on the sides of the sternum, later producing an absorption of the sternum and breaking through it. The edges of the sternum are so enlarged that they stand out about 1½ inches above the skin level.

The lesions that occur in this position can be divided into four classes. First: The lesions of the sternum itself, which never commence on the under side of the sternum—a myxomatous degeneration. There is first a softening of the bone, then an absorption, leaving a shell of bone, exactly as in cases of myxomatous tumor of the femur. It is a non-malignant swelling which finally produces an absorption of the bone.

Second: The lesions that occur in the cartilages. Third: The lesions that occur in the anterior mediastinum. Fourth: The lesions of the thymus gland. The types of tumors of the thymus are as follows:

TUMORS OF THE THYMUS

Normal adult gland, weight, 6 drams.

A. Sarcomata (54 cases reported to 1909).

1. Lymphoma	3
2. Lymphosarcoma	25
3. Sarcoma	10
4. “ globulare	7
5. Fibrosarcoma	2
6. Sarcoma macrocellulare	1
7. Unclassified	6

Age range, one year to fifty-eight.
Size: One ounce to 14½ pounds (De Silvestris).
Myasthenia gravis—Hassal's bodies in the muscles.

B. Carcinoma (11 cases).
    Epithelioma.

C. Mixed tumors.
    Cystadenochondrosarcoma.
    Peculiar thymus tumor.
    Hypertrophy.
    Epithelial degeneration of the thymus.

D. Non-malignant tumors.
  Lymphoma.
  Dermoid.
  Cystomyxoma.
  Epithelial degeneration.

Symptoms: Pressure on veins and nerves (rarely on arteries), trachea, esophagus, larynx (thymogenous dyspnea).

We collected the cases reported in the literature up to 1912. Up to that time there were reported 54 cases of the various types of sarcoma. The carcinomata are rare. Only 11 cases are on record. Sarcoma is about five times as frequent as carcinoma. These two types constitute the great bulk of tumors that occur in the thymus. The mixed tumors and the benign tumors occur less often. Let us review the embryology of the thymus gland. The origin of the thymus gland is from the third inner branchial cleft or third pharyngeal pocket. It is of endodermic origin. It is an epithelial structure. The connective-tissue elements are furnished by the mesodermic cells which surround the epithelial diverticulum and ultimately inclose and isolate its branching processes. The thymus reaches its maximum size at the age of two years. Then it remains stationary until puberty, when it rapidly degenerates,—that is, its epithelial elements,—only a mass of connective tissue remaining to indicate the location of the gland. However, the size of the gland is the greatest from the fourth month of gestation to the end of the third year of life, that is, its proportional size according to the weight of the body.

Considering the epithelial origin of the thymus, one would expect an epithelial type of tumor, a carcinoma, to occur most frequently, but sarcoma is far more common, and that type of tumor takes its origin in the connective tissue. If this is a tumor of the thymus, what is missing in the symptomatology? The symptomatology of thymus tumor is that of pressure on the trachea. There are no other special signs that we know anything about. If that mass is in the thymus gland, it should have compressed the mediastinum years before it did—before it came through the sternum. In this case it came through or around its border many years ago—so long ago that we cannot presume that it was then a malignant tumor. We cannot presume that this has been a malignant growth for twenty years. We must presume that this has been malignant only for the last year and especially so in the last four months. If the tumor originated in the thymus gland, she should have had a compression of the contents of the mediastinum. When you examine that patient, you see there is not the slightest bulging or pouching of the veins on the side of the neck. It is this bulging of the veins that tells you that the return circulation is embarrassed. Here it does not appear to be embarrassed at all. There is no evidence here of compression of the aorta, and at no time in the clinical course of this case has she suffered from dyspnea. That means that the growth of this tumor has been outward and not downward into the chest. The trachea and large bronchi have not been compressed. There is a limitation of the bone to the side, 1½ inches. That shows that the tumor began either in the bone or on the under surface of the sternum; that it is now a bone lesion that has started to grow outward. With a thymus tumor she should have dyspnea early; in fact, that is a pronounced early symptom. Palpation of the mass would almost lead one to believe that it was something that was in the sternum itself, but the history shows that the mass was noticed first on the left side, close to the margin of and as a part of the sternum; second, that later a second tumor appeared on the right side, with nothing in between the two tumors. They remained separate and distinct for years. Later the third mass appeared. The skin over this mass is discolored. It is a bluish red, shiny, and thinned out. The clavicles are palpable but not elevated.

Tumors that originate in the thymus gland do not materially compress the aorta. They are soft growths and never compress the aorta. They interfere with the other vessels, the veins, but not with the aorta. All these tumors press directly onto the vein, but never compress the aorta. If we attempt to remove that tumor, there is danger that we are going to open into the trachea and vessels beneath, if the inner portion has taken on the same character of growth that the outer portion has it should be adherent.

You can see from the skiagram that it is a type of tumor that resists the Röntgen-ray just as much as bone does. We know from this picture that this tumor is not an aneurysm. Why? Because if it were, all the tissue should have the same radiograph density, which it has not. An aneurysm gives a uniform shadow. Here is what aids me in differentiating between tumor and an aneurysm. Here is a portion of the tumor that has no blood in it and yet it has a density that obstructs the passage of the Röntgen-ray.

We will make an aspiration of this tumor and see whether it contains the grumous material that occurs in the cysts that are present in bone. The same type of material is found in the large lymphoid cysts in the thymus gland. The compact bone leads me to believe that it is not a thymus gland tumor. We will make the aspiration now. If this mass has bright red blood in it, then there is nothing to do. Then it is probably an aneurysm or an aneurysmal sarcoma of osseous origin, as there never has been an aneurysmal sarcoma of the thymus gland reported to my knowledge. If it is grumous blood, then we have the proposition to consider whether or not it is malignant. I feel that if it contains grumous blood, we are justified in operating. The chances are that when we attempt to take out the tumor we will get into both pleuræ. That is why we have the pulmotor here ready for use. If we get into the pleuræ, we will use the pulmotor to carry on respiration while we are doing the operation and then cover over the chest defect with a flap. On aspirating the left side of the mass we get bright red blood. That shows the vascularity of the mass. Now we will aspirate the other side and see if it contains fluid of the same character as the left side. If both these masses are of the same character, then we will know just what to do. There is not the slightest danger of hemorrhage from the aspiration. That mass has bright red blood in it too.

That shows that it is an aneurysmal tumor—not an aneurysm of the aorta, but an aneurysmal tumor. It is a tumor filled with blood. If I were to operate in this case, what would happen? The patient would die promptly on the table. We have still another hope. In the work that has been done on the thymus gland and thymus gland tumors there has been a considerable percentage of cures with the Röntgen-ray. The experiments show that these tumors have so little connective tissue in them that they contract under the Röntgen-ray. If you ask me what I think of this individual case, I would say that it presents the minimum chance of repair on account of the vascularity. I feel that she should have a systematic application of the Röntgen-ray. The treatment should be given with the magnesium plate interposed which shuts out the burning rays and just allows the penetrating rays to pass through the skin into the tumor.

I take it that it is an aneurysmal sarcoma of the bone and not of the thymus gland that has come through the bone and finally has come out under the skin in this way. Because it is of that variety is the reason why she has not had symptoms of pressure on the aorta, the pressure not being great enough to interfere with the ordinary circulation. That bright red blood means, I believe, that we are not justified in doing any operation whatever.

Let the record show that the tumor was aspirated on both sides and bright red blood withdrawn; that the needle was put in some distance under the skin; that it was found to be an aneurysmal sarcoma, and an operation was deemed inadvisable; that Röntgen-ray treatments were advised. The later course showed the necroses of skin and formation of a clot preventing hemorrhage. The skin destruction was from tumor invasion and not from the x-ray.

INFECTED BURSA OF OLECRANON; MINER'S ELBOW

HISTORY

PATIENT, male, aged fifty, enters hospital on account of pain and tenderness over the tip of the left elbow. April 22, 1913, he retired to bed at 7 A. M. after having worked hard all night shoveling copper ore. There was then nothing wrong with his elbow. At 11 A. M. he woke up with a dull, aching pain in the elbow over the olecranon. The pain increased in severity during the day, and the elbow was tender. He does not know whether his temperature was elevated. The next day the pain continued. The olecranon became swollen, and the tenderness on pressure increased. The swelling was aspirated and pus was withdrawn. On the following day a piece of tissue was removed from the elbow and examined microscopically. A report of sarcoma was made. Since that time he has had constant pain over site of incision, and it is increased on motion. He had no sore throat, cough, boils, or other local infections before the onset of the present trouble, and which might have caused a metastatic infection in the bursa of the olecranon. He remembers striking the elbow a sharp blow six or seven months ago, and that he had pain at the point of the elbow for about one-half hour afterward. He also states that the elbow is the seat of continual mild trauma during his work as a miner. He had typhoid in 1892.

Family history negative to tuberculosis and carcinoma. Venereal infection denied.

COMMENTS AND OPERATION

DR. MURPHY (Operation May 10, 1913): Where has he had pain since the operation?

INTERN: In the scar over the olecranon.

Dr. Murphy: That is important. He has no pain except at the site of the incision. That means that his elbow-joint is not affected. He has no pain in the elbow-joint at all. He has an unhealed wound over the olecranon which gives him pain when he flexes his arm. The lesion in this case is therefore limited to the olecranon—not the bone at all, but to the overlying soft tissues. And the particular seat of these infections is the bursa. Acute bursitis is usually the result of traumatism. The symptoms are a painful and tender, circumscribed swelling in the situation of the bursa, which fluctuates. Suppuration occurs as the result of infection through a wound. Chronic bursitis may follow the acute form, or result primarily from chronic irritation, as in the case before us. The most commonplace metastatic infection of this type is in the bursa over the patella—the prepatellar bursa. The clinical forms of involvement of this bursa are, first, the bursa known commonly as a housemaid's knee; second, an infection of the bursa forming a pus-sac; third, a condition known as hygroma. Whether this is a simple infection or whether it is a tuberculous infection matters little to the patient. He has pain and seeks relief. The ordinary case of housemaid's knee is usually the result of frequent kneeling. The knee at the patella is enlarged, white, and boggy to the touch. I remember one case in which I made an error in the diagnosis—I diagnosed the case as a tuberculosis of the patella. The swelling appeared to be a part of the patella, and all the signs and symptoms appeared to be those of tuberculosis. We saw it enlarge gradually and believed that it was a tuberculosis involving the patella. It was not a tuberculosis of the patella at all—it was a tuberculosis of the prepatellar bursa. I made an incision into it, the same incision as if I were going to remove the patella. On exposing the mass we immediately recognized it as a case of tuberculous housemaid's knee. We dissected out the infected prepatellar bursa and the patient got well promptly.

If the infection is of the chronic type, the bursa, after it has ruptured or has been incised, just continues to discharge. There is little or no tendency to healing. There is only one line of effec-

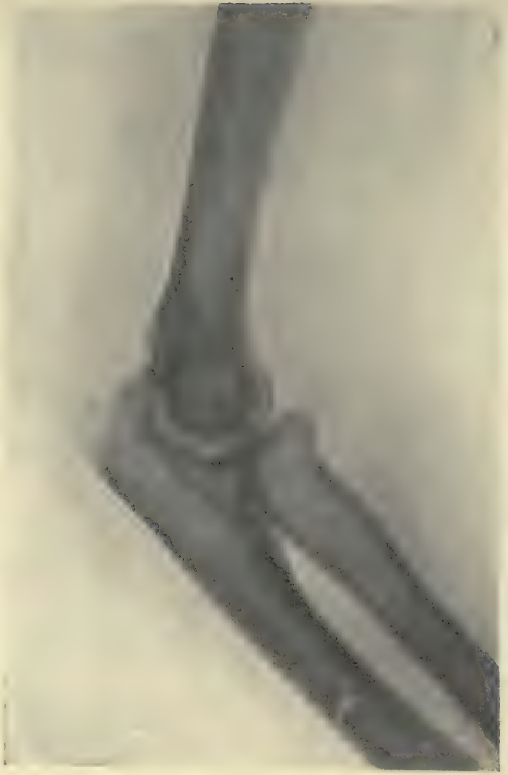

Fig. 216.—Olecranon bursitis. Skiagram made before operation, showing a slight enlargement of the tip of the olecranon and a rarefaction of the bone.

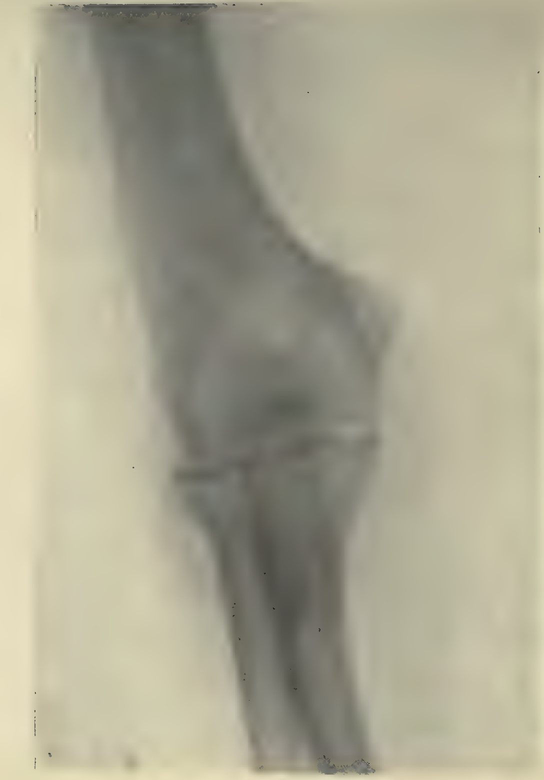

Fig. 217.—Anteroposterior view of same case. The small dark shadow over the tip of the olecranon marks the site of the infected bursa.

tive treatment, namely, complete excision of the sac, as in the case of a cyst.

Much more difficult conditions to handle are the infections that occur in the trochanteric bursa. They may come on suddenly, as this case did. Usually, however, they develop slowly. The swelling is most marked just behind the great trochanter. There are abduction and eversion of the thigh, but no limitation of motion in the hip. (See April, 1913, CLINICS, p. 337.) Finally some one cuts into it and makes a diagnosis of infection of the bone, takes off the tip of the trochanter, because the swelling is firmly adherent to the trochanter, and takes out the center of the sac that is readily accessible and permits the other portion of the sac to remain. What happens? It starts to fill again, and it is operated a second time, a third time, a fourth time, and still it grows until eventually it is believed to be a malignant condition. It is not malignant. It is merely an infection of the bursa. It sometimes extends five, six, or seven inches down the thigh, through the muscles in front and out behind the blood-vessels, up to the crest of the ilium, so that when you have made a complete dissection of one of these trochanteric bursæ, it seems as though the whole side of the patient has been opened. These bursæ continue to fill until every fragment of the sac is removed. A thorough and complete dissection must be made. The quantity of discharge is often great. In our last case of trochanteric bursitis the patient had a pyocyaneus infection on his arrival here and as much as a pint of pus was discharged a day.

The lesion in this case extends down to the periosteum. We will make a two-inch incision over the olecranon, retract the skin, and then carefully dissect out the infected bursa. We do not have to enter the joint at all. The old scar tissue will be excised, so that wound healing will progress favorably.

There is granulation tissue, which looks very suspicious. This may be a sarcoma, although sarcoma in this region is a very rare occurrence. We will make a microscopic examination of this tissue to determine whether it is sarcoma. Remember that he had this lesion cureted recently and the effort made by nature to effect a healing would account for the presence of this granulation tissue. The bone underneath this granulation tissue is compact normal bone. It is not soft bony tissue, but compact, which makes me believe that this is merely granulation tissue and not sarcomatous tissue. (This belief was confirmed by the pathologist who examined this tissue.)

We have removed all the sac. There has been practically no bleeding—no vessels to ligate. We will close the wound with a subcuticular catgut suture, which will obliterate dead spaces and take off the tension, and approximate the skin-edges with horsehair. The only dressing will be collodion gauze.

Let the record show that an incision was made over the olecranon and the old scar removed; that the bursa was dissected out and thoroughly extirpated.

[The wound healed per primam. The horsehair stitches were removed on the twelfth day. The patient left the hospital on the twenty-second day.—Ed.]

HEALED DUODENAL ULCER; CONSTRICTION OF PYLORIC ZONE OF STOMACH BY ADHESIONS—TUBERCULAR APPENDICITIS

HISTORY

Patient, male, aged fifty-seven, enters hospital on account of distress in abdomen, one-half to two hours after eating, which has existed for the past fifteen years. He has a sense of heaviness and fulness in the abdomen and belches gas, but has never been nauseated nor has he vomited. Bowels regular; never noticed any blood in the stools. He has not had tarry stools; no diarrhea. March 15, 1913, he had a dull aching pain in the right hypochondriac region, extending down to the iliac zone. This pain continued for four or five days. The attack was not accompanied by nausea, vomiting, or elevation of temperature. He was slightly tender over this region. He has never been jaundiced, and has not had ascites or edema of the legs or feet. He has lost 30 pounds in weight in the past fifteen years—since the onset of the trouble. Past history negative. He has had a left inguinal hernia for the last twenty years. It is easily kept in place with a truss. He had bronchitis in November, 1912. He has had a chronic cough for the last four or five years, with tubercle bacilli in the sputum. Family and venereal history negative.

COMMENTS AND OPERATION

Dr. Murphy (Operation May 1, 1913): This man had a tuberculosis some years ago—how many years ago, doctor?

Patient's Physician: Four or five years ago.

Dr. Murphy: What about the physical findings now?

Intern: There are no physical findings.

Dr. Murphy: What about the laboratory report?

Intern: Occult blood was found in the stools. Stomach-contents: total acidity, 33; no free hydrochloric acid; lactic acid present; no Oppler-Boas bacillus.

Dr. Murphy: This is a very interesting case. The symptoms are very indefinite, and we will reserve the diagnosis until we have opened the abdomen. Most of his symptoms point toward trouble in the right hypochondriac region, therefore we will open the abdomen through the right rectus muscle. The first thing we come across here is an adhesion on the anterior surface of the pyloric end of the stomach. I will first make an examination of the gall-bladder. It is not enlarged, but is whiter than normal. There are no stones in it, and it is compressible.

We do not see the stomach coming up into the wound as one usually does. Here is the adhesion on its anterior surface. There is the liver. It is normal. The stomach is buried under this broad, firm band of adhesions which is constricting it. We will bring the stomach out internal to that band and continue the examination. There is a thickened pylorus; in fact, a rather thickened stomach. Here is what I want to show you. It is a strangulating band extending completely across the stomach and constricting it in the pyloric zone. I never saw a similar condition before. There is where he has been having his retention. All the symptoms which he has had can now be interpreted. He has had blood in his stools, as we determined by examination, and it has been one of the positive conditions in every examination in his case. We will divide this band between two hemostats and ligate each end. See what happens when we free that adhesion! The stomach at once crowds up into the wound. That was a pathologic lesion. He has had an ulcer here at some time which caused that constriction. I never before saw a stomach constricted in this position by an external band. He had no food pain to speak of—some food distress, but nothing that one could call pain. He did not have a single symptom pointing to this condition. Dr. Mix had been over the case a number of times to see if he could arrive at a diagnosis. He failed. The presence of blood leads us to the belief that there should be an active ulcer somewhere in the stomach or duodenum.

The lesion we found here is pathologic, showing that at some time he has had a periduodenal infection and adhesion. Now I will examine the duodenum. It is dilated behind—the usual dilatation for the duodenum. Here on the under surface is the position for only a small percentage of ulcers. This portion is small and firm, but patent. Ninety-three per cent. of all ulcers of the duodenum occur in the first portion of it. There is no active ulcer palpable. He has an induration in the head of the pancreas not large enough to be interpreted as a carcinoma; he has a clean-cut induration in the under surface of the duodenum, which evidently followed some infection from his gall-bladder, and which accounts for its white color. There is also here the cicatrix of an old ulcer. See it slip out from under my palpating fingers!

The next place to look for an ulcer is in the first portion of the jejunum, which is not an uncommon location for ulcer. Picking up the large intestine and spreading it out, I am able to reach the first portion of the small intestine and bring it up. There it is, you see. There is the ligament of Treitz. On palpation I fail to find an ulcer there. Therefore an active ulcer in the upper zone of the intestine is out of consideration. Now we must locate the splenic flexure. He has no organic obstruction there. You know that at once—why? Because the bowel proximal to it is not dilated. The minute you see that normal bowel you know at once that there is no obstruction at the splenic flexure—obstruction is a more common condition in the splenic flexure with malignant disease than any other portion of the colon, except the lower sigmoid. The kidneys are palpable, and apparently normal in every way. Now we will examine the caput coli zone to see the condition of his appendix. He has a chronic lesion of his appendix, probably with ulceration, possibly a tuberculosis. He has not been particularly sensitive in this zone at all. The cecum is thickened; the meso-appendix is very thick; the appendix is hyperplastic. It is about one inch in diameter. I am convinced that it is a tuberculous condition.

First he had a pulmonary tuberculosis and then the appendix became involved. That accounts for the blood in the feces, although that is not a frequent symptom of tuberculous appendicitis. Now the clinical history and pathologic findings correspond. That is what they should do. The history of this patient demonstrated that he was in need of relief, and by surgical means. We were unable to make a diagnosis, even after most careful analysis. Therefore it was incumbent on us to advise an exploratory operation, to which this patient consented. You have to see these things to make them really yours. You cannot read them in books and make them yours. In removing this appendix I will have to amputate it very close to the caput coli in order to get an inversion of the stump. Where you have a tuberculosis, there is always the danger of a non-union. I never perform one of these operations without fear. The bleeding is so severe that compression with the forceps is necessary. The operation in these cases is the same as is done in all other cases of appendicitis—ligation of the mesenteric artery, purse-string suture in caput coli including the root of the meso-appendix in a double loop to insure hemostasis, crushing and ligating the appendix about ¼ inch from the caput coli, amputation of the appendix, cauterization of stump with 95 per cent. phenol, and inversion of stump with a double row of linen sutures. Has this man had tubercle bacilli in his sputum?

PATIENT'S PHYSICIAN: The last examination showed some; it was not a very active tuberculosis.

DR. MURPHY: Not very active. The disease was evidently arrested or quiescent.

Let the record show that the base of the appendix was enormously thickened; that we made the amputation close to the caput coli; that the inversion was easily accomplished; that for a circle an inch in diameter there was an infiltration of the entire wall of the caput coli. I have put in a few additional sutures above the purse-string so as to be sure to have a complete fixation. Why? Because this is a tuberculous process, and there is always the danger of non-union of the tissues. Remember the repair of tuberculosis is not by leukocytosis, as is the repair of suppuration; the repair of tuberculosis is by cicatrization. There is no infiltration or distention in the sigmoid or in the upper portion of the rectum.

That clears up the whole situation. The abdomen is closed in the usual manner—suture with eversion of the cut edge of the peritoneum; suture of the rectus aponeurosis; figure-of-8 silkworm-gut sutures, horsehair for the skin. Let the record further show that there was a strangulation or obstruction of the outlet of the stomach by a band of adhesions that passed clear across the first portion of the duodenum and permitted the stomach to fall down and loop over this firm band. This was the sequence of an old periduodenal infection. There was a thickening of the caput coli but no thickening of the ileum close to the caput coli. The appendix was tuberculous on microscopic examination. The prognosis is good. [The patient made an uneventful recovery. He left the hospital at the end of four weeks.—Ed.]

ANKYLOSIS OF KNEE WITH OLD FOCUS OF INFECTION IN TISSUES OUTSIDE OF KNEE, DISCOVERED AT OPERATION.—EARLY MANAGEMENT OF JOINT INFECTIONS TO PREVENT ANKYLOSIS.

HISTORY

PATIENT, female, married, aged thirty-three, enters hospital on account of inability to flex the right knee and slight limitation of motion in the left knee.

On the day preceding the onset of the trouble, in December, 1911, the patient was doing her housework, although she had a slight sore throat. In the evening of that day she sprinkled the lawn. Her feet became wet. The next morning she had a severe chill, followed by a high temperature. Three or four days later both knees and both elbows became painful and swollen. The swelling increased in degree and the knees were very red. The high temperature continued for six weeks. (Had no operations, no cast or extension.) During this time the least jar of the bed would cause severe pain. The right knee was more involved than the left. The elbows were not very bad. The swelling did not subside until May, 1912, and the patient was in bed all the time. On May 27th she was out of bed for the first time, but had to be carried around. The left knee was stiff, but not painful. The right was painful. The elbows at this time were all right. From that time on the patient used crutches and a cane and left knee limbered up, but the right became stiff and somewhat flexed. August 5, 1912, the right knee was straightened under anesthesia. She walked with a cane until November. February 15, 1913, the knee was again bent under anesthesia. She does not know what happened, but a cast was put on the leg. It had to be taken off at the end of two weeks because of swelling of the foot. She limped around one month with a cane. Since then she has not used it at all. At present she has no pain in the knee. The patella is fixed. Past history negative. Menstruation regular, twenty-four-day type, three days' duration, three to four napkins a day, no dysmenorrhea or leukorrhea. No intermenstrual or intramenstrual flow. Obstetric history: One child, normal delivery, nine years of age, healthy. No miscarriages. Family history: A brother died of tuberculosis.

COMMENTS AND OPERATION

Dr. Murphy (Operation May 3, 1913): What was the treatment of the right knee during the inflammatory process? Nothing was done; no cast applied; no extension put on. I say that merely to show that that is the regular way these cases are treated. What is the defect in that history? The questions that run in one's mind are, What was the cause of the multiple arthritis? What type of infection did this woman have that gave her the multiple arthritis? In order to form some estimate of the type of infection, you should know the duration of the preceding infection. A vital fact in connection with this case was not mentioned in that history. How long had she a slight sore throat?

Intern: I do not know.

Dr. Murphy: That is the defect in that history. If this were a streptococcic infection, she should get the metastatic deposit in the joints within twenty-four to forty-eight hours. If it is the type of streptococcic sore throat that we had in Chicago this last winter, which was so wicked, she should have the infection in her joints right away. Destruction of the joint is the sequence. If, on the other hand, it was the ordinary mild sore throat, a staphylococcus infection, then she should have had no destruction in the joint. If you get an idea of the primary lesion, then you can form an estimate of what type of infection occurred in that joint. In the streptococcus, grip, and pneumococcus cases the metastases in the various infections take place with as great regularity as do the eruptions in the eruptive diseases. You can tell in a second the time the metastases are due to occur. For instance, the streptococcus infections, as I said, always take place in the joints within twenty-four to forty-eight hours, sometimes earlier. The staphylococcus infection metastases never appear in the joint in that time; the pneumococcus infection metastases never appear in the joint in that time; the typhoid metastasis never appears in the joint in that time; the gonorrheal metastasis never appears in the joint in that time. The gonorrheal infection will metastasize into the joint between the eighteenth and the twenty-second day after the appearance of the discharge in the great majority of the cases. The pneumococcus metastasis takes place between the eleventh and the fifteenth days. Grip infection metastases in joints take place at about the same time as the pneumococcus metastases, and they are often associated with trauma. It is my conviction that every type of non-traumatic joint inflammation is a metastatic manifestation of a primary infection in some other portion of the body.

I like to cite one case in this connection in order to encourage observation in regard to the time of occurrence of metastases in the various infections in this class of cases. A doctor's wife had been sick for eleven days with grip. She had a purulent discharge from her nose and a purulent bronchitis. On the eleventh day she carried an ice-cream freezer on her left hip for a distance of some three hundred yards. That night she had a chill and pain in her hip. She did not get out of bed for twelve or fifteen weeks; when she did, she had a bony ankylosis of her left hip, the result of the metastatic grip infection precipitated by the trauma. I merely cite this case as an example, because these cases are all very much alike. In the present case the doctor neglected to get the date of the beginning of the sore throat, so that we are unable to form an estimate as to when the joint lesion began in relation to the primary infection. We know that she had a multiple arthritis. Her joints were inflamed, and severely inflamed. While she was in bed nothing was done to counteract the muscular contraction always present in these cases. The limbs were permitted to be flexed and the result of this muscular contraction was that the articular ends of the bones of the knee-joint were pressed together tightly. The articular cartilage became the seat of a pressure necrosis and a fibrous ankylosis took place. Then she was anesthetized and the limbs were straightened. These are the two conditions which favor the occurrence of ankylosis.

It is important for us to learn to differentiate the treatment of the two great types of lesions that occur in joints. One is the tuberculous type and the other is the metastatic infectious type. Tuberculosis causes less destruction and tends to repair when the joint is completely immobilized, and with less likelihood of ankylosis occurring. Why? Tuberculosis heals by encapsulation of the infective focus, not by the action of the phagocytes and leukocytes. The metastatic infections heal just the other way—not by encapsulation, but by the action of the leukocytes—an immunizing process. If you immobilize an acutely inflamed joint for weeks, you insure the occurrence of ankylosis. Nothing else can be expected. What was the first management of this case from a medical standpoint? Nothing was done. She was kept in bed. That was all. Every acute arthritis, multiple or single, that commences with a chill, is a surgical lesion. What do I mean by that? I mean that if it begins with a chill, it is probably going to result in a destruction of one or more joints unless proper treatment is instituted. The chill is the differentiation between the type of infection that repairs without the destruction of the synovial membrane and joint cartilages, and the type that tends to destroy the synovial membrane and cartilages, and causes a bony ankylosis. It is the same process in every one of these cases. Let us sum up the treatment:

First: The limb must be kept in a straight position with an ordinary Buck's extension applied, enough weight being attached to overcome muscular contraction—20 to 30 pounds. Do not put a 6- or 10-pound weight on a 250-pound man. Put on enough weight to separate the articular surfaces and keep them separated and overcome the contraction of the muscles. Keep the limb in the best position to avoid the ankylosis. If you cannot avoid the ankylosis, you at least can keep the limb straight, and later

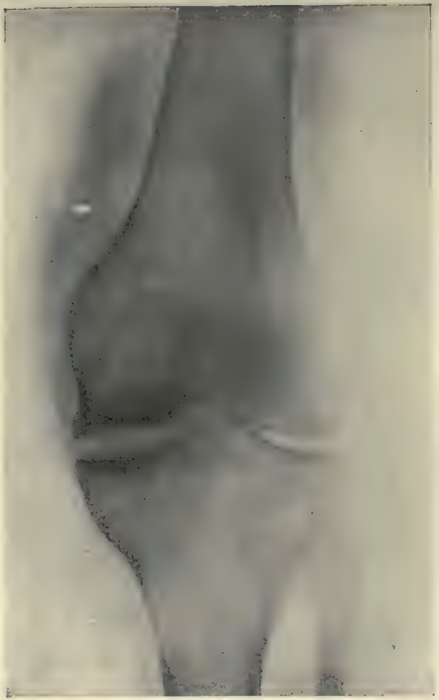

Fig. 218.—Anteroposterior view, taken before operation. The internal condyle appears to be thicker than the external, but this is due to the shadow of the patella, which was united to the femur by a bony ankylosis. The joint cavity between the internal condyle and the internal tuberosity is obscured by the deposition of dense fibrous tissue. This was not a bony ankylosis between the femur and the tibia.

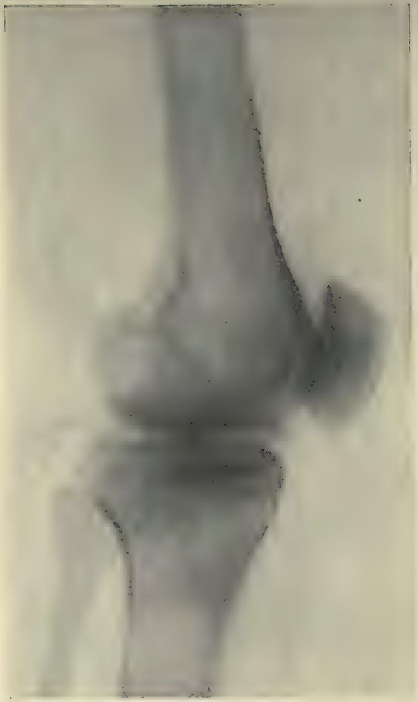

Fig. 219.—Lateral view of same case, showing how the patella has united with the femur.

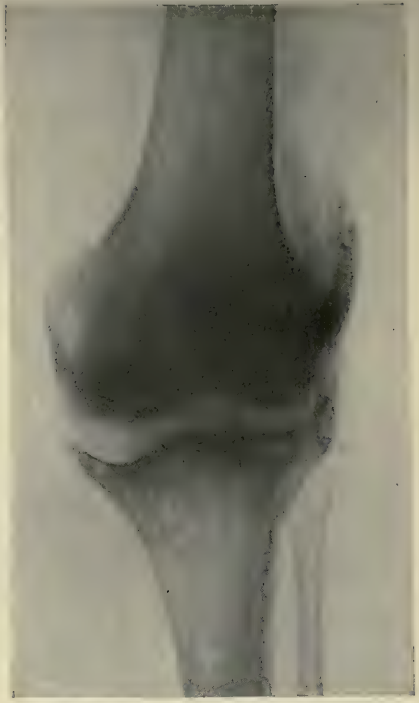

Fig. 220.—Skiagram, anteroposterior view, made about ten weeks after the operation. The joint cavity is intact. The patient had voluntary motion in the knee. The articular surface of the tibia has been well reformed, and the leg is plumb.

you can do some operative procedure for the production of a new joint.

Second: Relieve the intra-articular pressure due to the accumulation of infective products in the joint by aspiration—not incision and drainage, but by aspiration.

Third: Render the contents of that joint a poor culture-medium by injecting into the joint some antiseptic which will also coffer-dam the lymph-spaces around that joint and stimulate a chemical inflammatory reaction. You can increase the number of polynuclear leukocytes in the joint contents by the injection of a 2 per cent. solution of formalin and glycerin, which must have been mixed at least twenty-four hours preceding use, as we have demonstrated again and again. That produces a polymorphonuclear leukocytosis, a coffer-damming of the lymph-spaces, and renders the joint contents a poor culture-medium for bacteria. This is not an ideal solution to use. We are going to have a better remedy some day, one which we can inject into the blood in the early days of the disease. It is going to be a chemical remedy.

Thus far we have treated the condition locally. We have done all we could to check the disease and to prevent the further destruction of tissue and the consequent occurrence of deformities and loss of function. This treatment will not restore destroyed tissue. It merely prevents further destruction.

Fourth: You avail yourself of the aid you can obtain from autogenous vaccines, which is very great in many of the cases. It is one of the things which none of us can afford to scorn. The autogenous vaccine is far more valuable than the stock vaccine. (See article on Vaccine Therapy in August, 1913, CLINICS.)

That is the management of the early stage. It is not the treatment for the patients who come to us because of a fibrous or bony ankylosis representing the final stage of arthritic pathology. We feel that we have worked out a very good plan for the cure of these cases of ankylosis.

On examination of the skiagram of this case we find that this is not a complete bony union, but is a fibrous ankylosis between the tibia and the femur. However, a fibrous ankylosis gives her much more trouble than the bony one would. I would rather have a solid bony ankylosis, because I can get very much better results from an arthroplasty with that type of ankylosis than I can when they come in in this condition. There is, however, a bony ankylosis between the patella and the femur.

VISITING DOCTOR: How long do you keep up the extension in these cases of arthritis?

DR. MURPHY: These patients are kept in bed three weeks. If the inflammation in the joint has subsided at that time, they are allowed to sit up in bed or even on a chair. We keep on the extension at night for three weeks more, but the length of time depends entirely on the progress of the case.

[Here Dr. Murphy detailed the results of a number of cases of ankylosis, and after outlining the post-operative treatment of hip arthroplasties, the following question was asked.]

VISITING DOCTOR: Do you always dress these cases with the legs in abduction?

DR. MURPHY: Yes, always. We dress them with the Rainey travois splint; that is, we place both limbs in an abducted position. Remember you cannot abduct one limb. The tendency of the adductor group of muscles is to contract. If you do not dress both legs in the abducted position, you will find that the patient has twisted the pelvis so as to get into a comfortable position and the operated limb will be straight or even adducted to the pelvis. By keeping both legs abducted you prevent that change of position. The rule is that when these patients come to us, the affected limb is flexed and adducted. Why? Because the tendency is to draw up the leg to relieve the pain in the joint as much as possible, and this change of position has a similar effect to extension in the relief of joint tension. We demonstrated that in some experimental work done by Dr. Speed on the cadaver. When the hip-joint was injected, the limb always was rotated inward, adducted, and flexed at the hip. The capsular ligament did not rupture until the intra-articular pressure had reached 22 pounds or upward to the square inch. In the case of the knee-joint the pressure went as high as 120 pounds before the capsule ruptured and the knee was semi-flexed. That shows the enormous tension under which the products of infection may be held in the hip and knee before nature gives relief by rupture of the joint-capsule.

When there is a destruction of the synovial membrane and joint cartilage, with the formation of a fibrous ankylosis, it is useless to tear up the adhesions by forcible flexion under anesthesia because there is always a return of the ankylosis, with a greater fixation of the joint than before. We treat these cases of fibrous ankylosis just as we do a bony ankylosis—by arthroplasty, interposing a pedicled fat and fascia flap between the ends of the bones.

We will make the usual exposure of the joint by means of two incisions—one on either side of the patella, extending from one inch above to two inches below the patella. The incisions curve away from the patella above, so that the upper extremities of the incisions are separated 4 inches, the lower 2½. These incisions extend down to the outer layer of the capsule of the joint. The edges of the incisions are undermined and retracted. Ah! here on the outer side of the joint, in the deep fascia, is a focus of infection about the size of an English walnut. That is too bad! There was no evidence of the existence of such a focus before operation. That will interfere materially with the subsequent course of this case. [On microscopic examination the pus was found to be sterile. It contained many lymphocytes and leukocytes, but no bacteria.] We will swab this zone thoroughly with lysol solution.

You will note that there is very little bleeding here, even though no constrictor was placed on this leg. Next we prepare the flaps—an external and an internal flap—to be interposed between the femur and the tibia. The base of each flap is down over the inner and outer tuberosities of the tibia respectively. The flaps measure about three inches in length and about 2 to 2½ inches in width. We must have enough tissue to cover all the articular surfaces of these bones without tension on the flaps. We now separate the adhesions in the joint. In order to give room for these flaps and to avoid pressure on them, we will remove almost one inch of the upper end of the tibia with a large curved carpenter's chisel—preserving the normal anatomic conformation as much as possible, so that the leg will be plumb and no tendency be shown later to adduction, abduction, or subluxation of the tibia.

We lose quite a little blood in this operation, but there is a definite reason for that. If we put on a constrictor, when we remove it at the conclusion of the operation the oozing continues and a hematoma forms. One of the most dangerous conditions in connection with this work is the formation of a hematoma. That is what we fear most. We have had several such cases and the subsequent smooth progress of the cases has been delayed very much. We find that the cases in which hematomata are most apt to form are those due to tuberculosis or where there has persisted a hidden focus of infection, as in this case. We had a case very similar to this not long ago. The result was not satisfactory. The patient got a straight limb, but there was not sufficient motion. Pus in the joint always hazards the result greatly if the pus is still virulent. It may not be. It may be dead pus that has been there for a long time and which does not contain viable microörganisms.

Let the record show that there was a small circumscribed pocket of pus outside of the joint in this case.

Now our flaps are ready. There is only one small ligament on the external surface of the joint. That runs from the tip of the fibula up and across the joint to the external condyle of the femur. That ligament is usually not disturbed, because it is not in the field of work. Before interposing these flaps we will direct our attention to the patella. The patella is fixed to the femur as we estimated from the skiagram. It is fixed only over the external condyle, however. I will free that patella with a straight chisel and retract it out of the way so I will be able to get into this joint fairly well. We will rotate this patella later, turning it turtle, so that the ankylosis will not recur. After this rotation the prepatellar bursa will be the new patellar lining for this joint.

In removing the upper part of the tibia we are very careful not to injure the contents of the popliteal space,—the popliteal artery, vein, and nerve,—and during every step of the operation we guard against traumatizing the external popliteal nerve as it winds around the head of the fibula. We do not interfere with the posterior layer of the joint-capsule because that must be kept intact to prevent subsequent subluxation of the tibia. We have removed the bone, now, reconstructing the tuberosities and the intercondyloid ridge so that the leg will be perfectly plumb.

Now we are ready to interpose our flaps. We draw the inner flap into the joint and suture it to the stump of the posterior crucial ligament and the posterior layer of the joint-capsule behind and to the periosteum beneath the patellar tendon in front. Just two tacking sutures hold that flap in position. Then the outer flap is drawn into the joint, overlapping the inner. It is also fastened in place with two sutures.

We have been very much surprised in these cases to find how little support is given the knee-joint by the lateral ligament, and that it has no support at all from the capsule until the normal anatomic range of motion is exceeded.

Note that the oozing has ceased. We have not had to ligate a single vessel. See how these interposing flaps overlap—nearly an inch. All the raw bony surfaces are covered by these fat and fascia flaps. The nutrition for both of the flaps comes from below. Now our flaps are fixed. The tendency is to have the leg a little bit adducted. That gives the best sequence.

Now we will turn the patella, making a rotation of 180 degrees. By means of small sharp hooks that is accomplished without great difficulty. By that rotation we get a new lining for the joint and have assured a cure for the ankylosis of the patella. We will trim off the edges of this patella so that they will not press against the skin. That makes practically a normal patella. In order to maintain the patellar rotation, we suture the edges of the quadriceps tendon and ligamentum patellæ to the underlying fascia by means of chromicized catgut. Remember that the outer margin of the patellar portion quadriceps tendon is now on the inside, and likewise of the ligamentum patellæ. That patella is fixed on both sides so that it has to stay in the midline. That keeps the patella in the intercondyloid notch all the time.

You can see how difficult it is to do that. No one can read of it and do it. You have to see it in order to have a mental picture.

Let the record show that there was a bony ankylosis between the patella and the femur; that this was chiseled free; that there was a fibrous ankylosis between the tibia and the femur and that this was freed; that the usual arthroplasty was performed.

We are now ready for the closure. The first step is to take the tension off these skin-flaps so the margin of each flap is free from tension. I do that by means of subcuticular chromicized catgut sutures. That also helps to give support to the joint. If you do not get all the tension off the skin, you will have a pressure necrosis. That would be serious. Picking up each flap in that manner, we also obliterate the dead space and lessen the likelihood of a hematoma occurring. We have a thick skin-flap left, notwithstanding the fact that we had a good-sized interposing flap. The skin-edges are approximated with horsehair; the wounds are dusted with bismuth subiodid and sealed with collodion gauze. A large alcohol and 1 per cent. phenol gauze dressing will be applied and the leg placed in a wire cage with a 15-pound Buck's extension applied.

Let the record show that there was a small circumscribed abscess on the outer side of the articulation; that this was opened and the smear showed lymphocytes, polymorphonuclear leukocytes, and squamous cells but no bacteria; that this was cleaned with lysol solution; that there was a fibrous ankylosis between the tibia and the femur which was freed and a bony ankylosis between the patella and femur which was chiseled free; that the internal and external tuberosities of the tibia were chiseled off and the usual fat and fascia flaps inserted and sutured over the tibia; that the patella was rotated 180 degrees and fastened; and that the wound was closed with horsehair.

[The patient did very well at first. The stitches were removed on the fifteenth day, with primary wound healing, notwithstanding that there was a considerable accumulation of blood beneath the flaps. A week later a discharge, bloody but aseptic, broke through the skin in the lines of incision. This continued for several weeks. The wounds, which had been opened all this time and remained sterile, were sutured with silkworm-gut and healed promptly. These stitches were removed after two weeks. Then the outer and lower edge of the patella produced a pressure necrosis of the skin, followed by a necrosis of the patella. This was cureted and the skin-edges approximated with adhesive plaster. The wound has not healed yet. The patient has about 20 degrees of free voluntary motion in the joint and will, I feel sure, have very good motion in the end.—Ed.]

CONGENITAL IDIOPATHIC DILATATION OF THE COLON; PARRY'S DISEASE; HIRSCHSPRUNG'S DISEASE

HISTORY

PATIENT, female, aged ten, enters hospital complaining of chronic constipation. There are frequent periods when the bowels fail to move for a couple of days, and then only after heavy catharsis and enemas. She has taken olive-oil enemas daily practically all her life. Sometimes she does not need cathartics for three or four months, but for the past three months she has been taking laxatives daily. The trouble dates back to birth when the bowels failed to move for twenty-four hours and feces had to be removed with a catheter. She did not have a normal bowel movement until eight months old, and then stools were the size of a match. At present the stools are the size of a lead-pencil and flattened. When six months old, she began taking daily enemas of olive-oil, and cathartics when needed, which was about half the time. When four years old she had a bad attack of obstipation; no bowel movement for over a week, jaundice, and a failure of the kidneys to work. The left side of the abdomen has always seemed a little larger than the right and is occasionally tender and painful.

Past History.—Whooping-cough at six years; chicken-pox at seven years. Has two brothers living and well. One sister died of intussusception when nine months old. The patient has never been able to eat anything containing seeds, and always eats light evening meals. Has always been troubled with gas in bowels, and it is hard for her to pass it. A doctor who once examined her when she was quite young said she had a stricture of the large intestine.

COMMENTS AND OPERATION

DR. MURPHY (Operation June 4, 1913): That is a good description of the condition present. It is a congenital idiopathic dilatation of the colon. It is known as Hirschsprung's disease, but should be called Parry's disease, because he described this condition sixty years before Hirschsprung did. I have placed an outline on the board of the etiology, symptoms, and treatment of megacolon, because it aids us in getting a mental picture of the conditions that lead to the present pathologic situation.

CONGENITAL IDIOPATHIC DILATATION OF COLON

(Parry's disease (1825); Hirschsprung's disease (1886))

Etiology
  (*a*) Mechanical
  (*b*) Physiologic
  (*c*) Neuropathic

Symptoms
  (*d*) Congenital.
  Obstipation.
  Emaciation.
  Intoxication.

Area involved: In 15 per cent. the entire large intestine is involved. The sigmoid is practically always involved.

Treatment
  (*a*) Colopexy.
  (*b*) Coloplication.
  (*c*) Permanent colostomy.
  (*d*) Entero-anastomosis.
  (*e*) Resection of dilated segment.

The skiagrams of this case are very convincing. You will note that enormous dilatation in the left side of the abdomen. That is the sigmoid zone. We gave this patient bismuth and made a series of pictures. Then we injected two quarts of bismuth fluid into the large intestine. There is an enormous dilatation of the large intestine, so that we are convinced that this is a case of Parry's (Hirschsprung's) disease. When he described this lesion first, he described it as a congenital dilatation. That is a

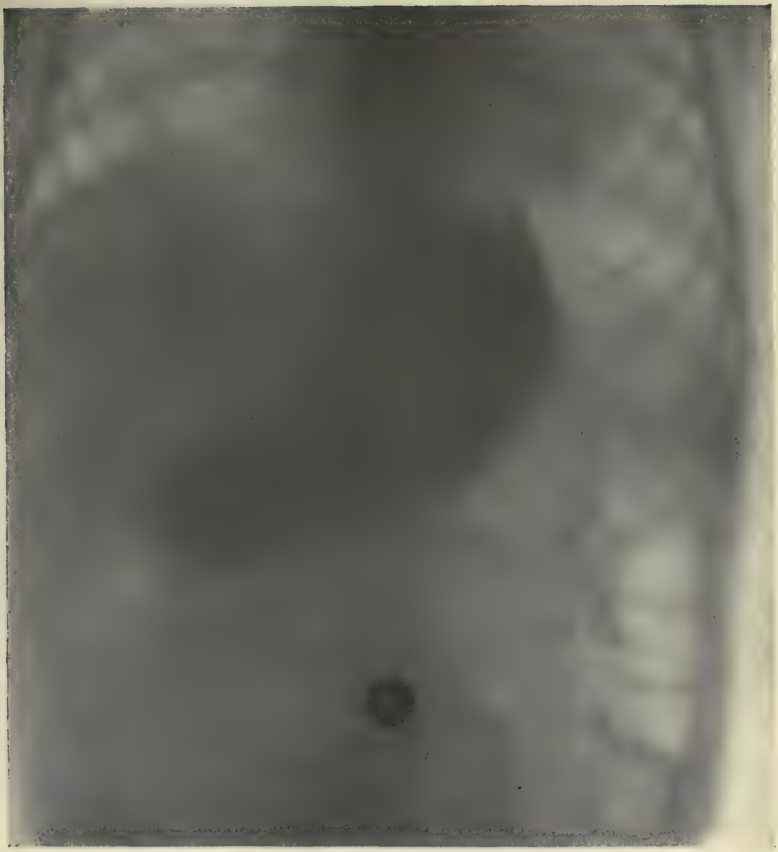

Fig. 221.—Skiagram made immediately after the ingestion of the bismuth meal, showing the outline of the stomach only. The lighter areas on the right are thrown by the gas in the dilated large intestine.

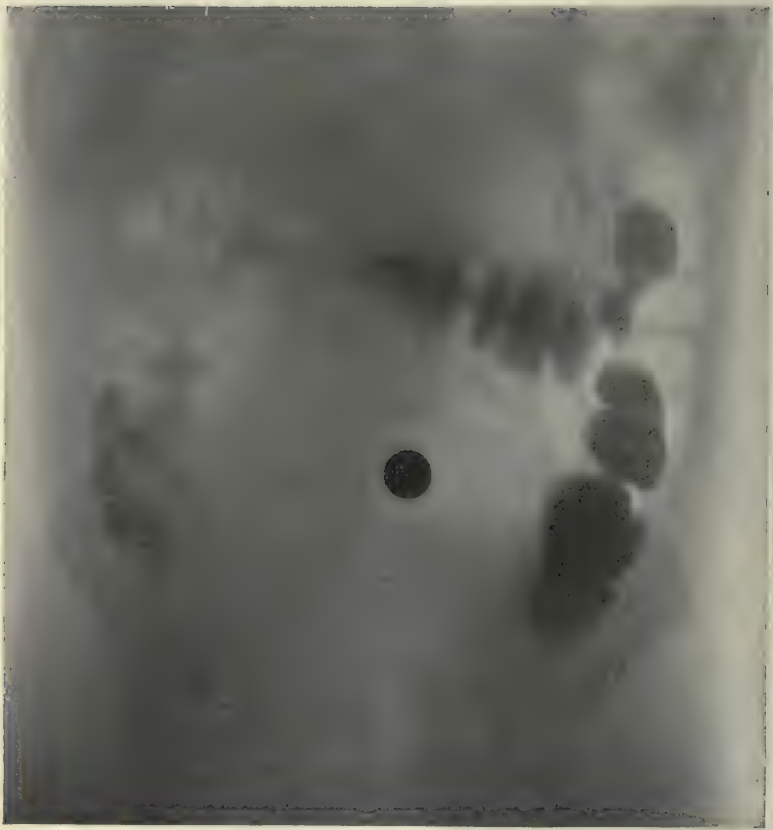

Fig. 222.—Skiagram made twenty-four hours after the ingestion of the bismuth meal. The bismuth has not only left the stomach, but also the small intestine. It is now in the large bowel, from the cecum to the sigmoid. The bowel is apparently in normal position. There is a large, light shadow extending from the pubes up into the pelvis, and just to the right of the umbilicus. This is the dilated portion of the rectum and the sigmoid.

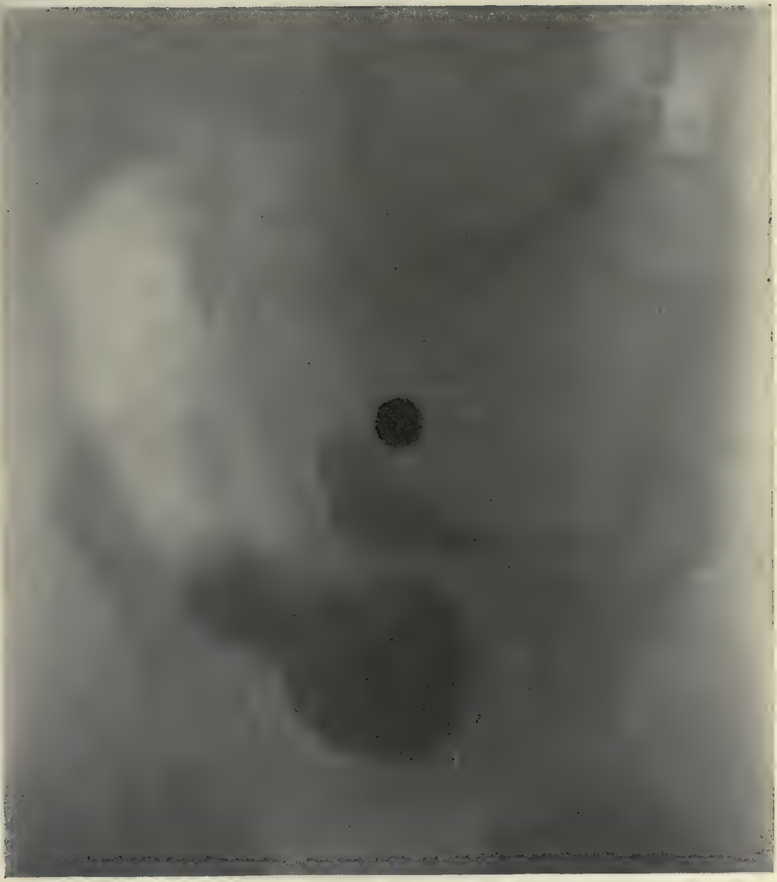

Fig. 223.—Skiagram made forty-eight hours after the ingestion of the bismuth meal. The bismuth has disappeared from the ascending colon, but may still be seen in the transverse colon, extending to the right of the spinal column and up to the splenic flexure. The descending colon appears to be embedded, but is dilated. The sigmoid contains some bismuth, the rectum a great deal of it. The bismuth is evidently lodged in a pocket above the constriction in the rectum.

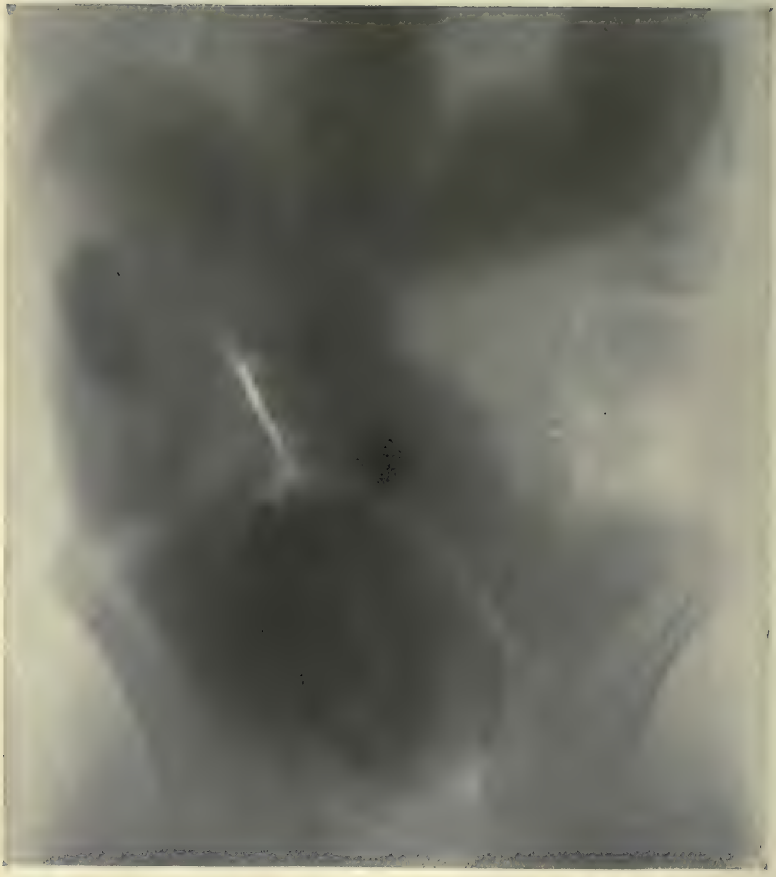

Fig. 224.—Skiagram made immediately after the injection of the bismuth per rectum. The large bowel is filled with bismuth, and obstructs all the other organs in the abdominal cavity. The degree of dilatation is well shown in this picture.

true Hirschsprung's disease. The condition described under the name Parry's disease was merely an enlargement of the intestine. Hirschsprung's disease is usually met with in young children, and is characterized by obstinate constipation from birth, with distention and enlargement of the abdomen. The anal canal and rectum are usually normal, the dilatation beginning in the lower part of the pelvic colon. Usually the pelvic colon alone is affected, but it may extend to the descending colon, the transverse colon, and even to the cecum. The wall of the affected bowel is greatly thickened, principally because of the enormous hypertrophy of the circular muscular coat. The causes are as outlined on the board. In this case I believe we have a mechanical condition: first there was an obstruction near the anus and then a dilatation.

We will do an exploratory laparotomy here and endeavor to determine just what line of treatment should be adopted. We will open the abdomen through the left rectus muscle in the usual manner. We incise this peritoneum with very much more caution than we do in the average case because of the distended bowel. We grasp it with two tissue forceps and incise it on the slant between these forceps very carefully. The bladder in these cases may extend high up, so that we must watch for it also. We have opened the peritoneum without mishap. The urachus is in view. The bladder appears to be displaced high up—several inches higher than it should be. I enlarge this incision downward to examine the lower bowel as it is in the abdomen. We do not want to take that dilated bowel out. I will see if I can find, by the Trendelenburg position, where the obstruction is and what it is. I palpate the sigmoid. It is negative. Next I come to the rectum and I find something to account for this condition.

The obstruction is in the first portion of the rectum, one of the places I feared it would be and one of the places that is the most difficult of all to handle. There is the bladder. I told you about the bladder being close in my field, right at the line of division. If I did not know this, I would have been right on or into the bladder. That is one of the precautions one takes when he has one of these anatomic anomalies. I want to get a view of the situation. See the obstruction there! It is an annular band in the first portion of the rectum, close to the junction of the first and second portions of the rectum at the uterovesical fold of the peritoneum. I will now retract the intestine out of the field so as to make a full inspection and then I will decide on the line of procedure.

An anastomosis cannot be made below because the obstruction is beneath the peritoneal fold of the rectum. The plan of procedure will be to close this abdomen and go in from below. The area involved in the distention is all of the large intestine, but not the small intestine. I am right at the bladder. I am exercising the greatest care not to cut the bladder. I will show you the small intestine. It is normal in appearance. It is a dilatation of the large intestine. The fibrous striæ of the large intestine become obliterated by the dilatation so that this portion of the bowel resembles the small intestine on its surface, but not in size. I see nothing on its surface to indicate that it is the large intestine. We know that it is the large intestine because we can trace it from rectum to cecum. The enormous thickening of that bowel is probably due to the effort of the musculature to overcome the obstruction during all the years of her life, and that in a secondary hyperplasia with dilatation.

Now that we know where the obstruction is, we know just what we have to do. I had hoped that the obstruction would be high enough up so that I could do a transposition of the ileum into the large intestine below the point of obstruction. If I could have done that, I would have side-tracked the large intestine completely, but that was impossible.

Let the record show that Sister reports the sponges counted and correct.

We are ready for the closure of the abdomen. We will have a great distention of this abdomen immediately after this operation, so that we are putting in an additional suture, using the chromicized gut. That will last a little longer than the plain catgut. The peritoneum, the rectus aponeurosis, and the skin incisions are closed in the usual manner, and without a drain.

Not long ago we had a child that had some 12 inches of the sigmoid and descending colon involved in dilatation it was circumscribed and was a true congenital megacolon. We made a complete intestinal resection and an end-to-end union. The zones above and below were not dilated. Here the bowel is dilated a long distance above; that is what makes me believe that it is purely an obstruction lesion. The dilatation of the large intestine in this case is the sequence of continued obstruction. When you come to estimate the mechanics of this condition it is necessary to take into consideration the degree of pressure by the square inch where this obstruction is. The diameter of the bowel at the point of obstruction is not over 1/4 inch, possibly 1/3, the diameter of the intestine up where it is distended is easily 3 inches. To get the comparative total pressure take the square of the square of the circumference; *i. e.*, is as $\frac{1}{9}$ to 9 or 81 times as much. The pressure is enormous. Think of the weight and pressure that are carried by the large intestine above the obstruction! That is why you have these conditions getting worse and worse unless you relieve the obstruction. The degree of distention varies greatly. The periods between the bowel movements vary between a few days and a few months. These patients have gone as long as sixty, sixty-five, or seventy days without a bowel movement. We had a patient who had 56 pounds of feces in the large intestine!

Of all these etiologic factors, physiologic, neuropathic, mechanical, and congenital, to my mind it is more often a congenital condition than anything else. This was, I believe, either primarily a congenital dilatation or more likely it was primarily a fibrous stenosis of the intestine. Occasionally, high up in the intestine, we find these stenoses at the point of attachment of the vitello-intestinal duct; that is, 39 inches above the ileocecal valve. You rarely ever have great dilatation of the intestine above that point. It is at this point that we so often have a diverticulum. The constipation is the first symptom. It is also a continued symptom. Then comes the dilatation of the bowel and of the abdomen. Finally comes the emaciation that follows the intestinal intoxication, the fecal intoxication, the dry skin, the fetid breath, headache, and all the symptoms of that condition.

There would be no advantage in short-circuiting the ileum in this case and throwing it into the sigmoid, because the obstruction is lower down below the point of peritoneal covering. If we could go below the obstruction, then it would be an entirely different proposition.

Of the treatments that have been suggested for Hirschsprung's disease, the first is colopexy. Of course, that is out of the question here because the condition involves the entire bowel length. This is a congenital stenosis, not a typical congenital Hirschsprung's dilatation. Some eight or ten years ago we resected 20 inches of an enormously thickened intestine with a comparatively normal intestine below and above this zone with no obstruction at all, showing that this is not always an etiologic factor in these cases. A colostomy would have no value at all. A permanent artificial anus would not relieve the situation a particle and materially increase the discomfort. We are able to reach this obstruction from below. We will dilate the stricture by force under anesthesia at first, and finally put in a permanent plug, just the same as you do in a stricture of the male urethra. If you are dealing with a urethral stricture, you often have difficulty in getting the filiform bougie in at first, but when you get it in be sure that you leave it in; put in another and still another, but do not take out the primary one, because if you do, you won't get it in the second time. I recall one of my very early experiences with the late Dr. James Nevins Hyde, when I knew comparatively nothing about the urethral dilatation compared with that eminent author. He put in his filiform bougie in one of these cases, and took it out to put in a larger one. He failed and was relieved from the case. I put in one and that dilated the urethra. I waited, and at the end of five days I could put in a No. 6 catheter with ease. That is just what happens with all strictures of this type. If you will leave a dilator of some kind in place, absorption of the stricture—the cicatricial tissue—takes place and finally you have a complete dilatation. I believe we will be able to dilate the stricture in this case so that we will be able to carry the case to a favorable termination. The operation we did this morning was purely an exploratory laparotomy.

Three weeks later under anesthesia we forcibly dilated with the finger a stricture three inches above the sphincter and started the use of the bougie.

[Rectal dilators of increasing size were used daily for twenty-five minutes at a time, and after five weeks the patient's condition had improved so much that she was permitted to leave the hospital. She returned for examination July 19, feeling better than she ever had. She had a normal bowel movement every day and a lower rectal lumen larger than normal.—Ed.]

ANKYLOSIS OF HIP FOLLOWING SORE THROAT; METASTATIC ARTHRITIS; ARTHROPLASTY

HISTORY

PATIENT, male, aged seventeen, entered hospital May 1, 1913, on account of absence of mobility in the left hip. June 28, 1911, he had a sore throat and earache. He went swimming every day until June 30th, when he went to bed because of the severity of the earache. The morning of July 2d "something popped" in the ear. There was no external discharge. That afternoon, on trying to get out of bed, he had severe pain in the right hip. That night he had a temperature of 104.5° F. He had no chill preceding the onset of the pain or fever. July 3d he was taken to a hospital. His temperature was still high—103° to 104° F. That night an incision was made on the anterior surface of the left thigh; the femur was exposed and chiseled. No pus was found, but a drainage-tube was inserted. There was no discharge from the tube, however. The temperature remained elevated and the boy became delirious. He tore off the dressings and pulled out the drainage-tube July 5th. At this time there were redness and extreme tenderness over the trochanter, and he complained of pain about the anterior superior spine of the ilium. The incision in the thigh healed in two or three weeks. Temperature elevation and pain continued during July and about the first of August the hip-joint was aspirated. The result was negative. One week later the "joint" was injected with formalin in glycerin. By this time the leg was drawn up on the thigh and the knee was partially flexed. During August the temperature remained high. It would reach 103° F., remain there for seven or eight days, drop to 100° F. for two or three days, then rise again. By the first of September the boy was very much emaciated and had severe bed-sores. The first week in September the leg was straightened under anesthesia and extension applied with a cast extending from the ankle up to and including the hips. This cast was kept on six weeks, until the middle of October, during which time the pain in the hip became more severe. Temperature continued as during August. The middle of October the pain was so severe that it was decided to remove the cast. When the cast was removed, much pus and serum were found in the cast. There were three sinuses—one at the upper end of the incision, one in the groin, and one near the trochanter. The temperature dropped immediately, but the discharge continued for four or five weeks, until the twentieth or twenty-fifth of November. The ankle, knee, and hip were stiff. Mobility returned in the knee and ankle, but the hip remained stiff. December he spent at Hot Springs, Ark. He gained 21 pounds in weight in twenty-three days. He does not remember having any pain in the hip since December, 1911. He used crutches until January 14, 1913. Now walks unaided by cane or crutch. At present the hip is painless, but without motion.

COMMENTS AND OPERATION

Dr. Murphy (Operation May 3, 1913): It is the same story that one hears in so many of these cases—a primary infection in the throat (pharynx, not tonsil), with a metastatic infection in a joint. If this had been an osteomyelitis, the treatment given—exposure and chiseling of the femur—would have been the proper one, but this was a metastatic arthritis, not an infective osteomyelitis. What had he been doing the day preceding the onset of his trouble? He had been swimming, not only that day, but for several days, even when his throat was sore; and he may have sustained some trauma that boys often get while in swimming which could precipitate or focalize the infection in the hip. The leg is flexed slightly at the hip and adducted. That is the usual position of the leg when a bony ankylosis is present in the hip. Motion range is normal in the knee and ankle. The skiagram shows that there is not a complete bony ankylosis over the whole surface of the joint. Only the lower portion of the head is in-

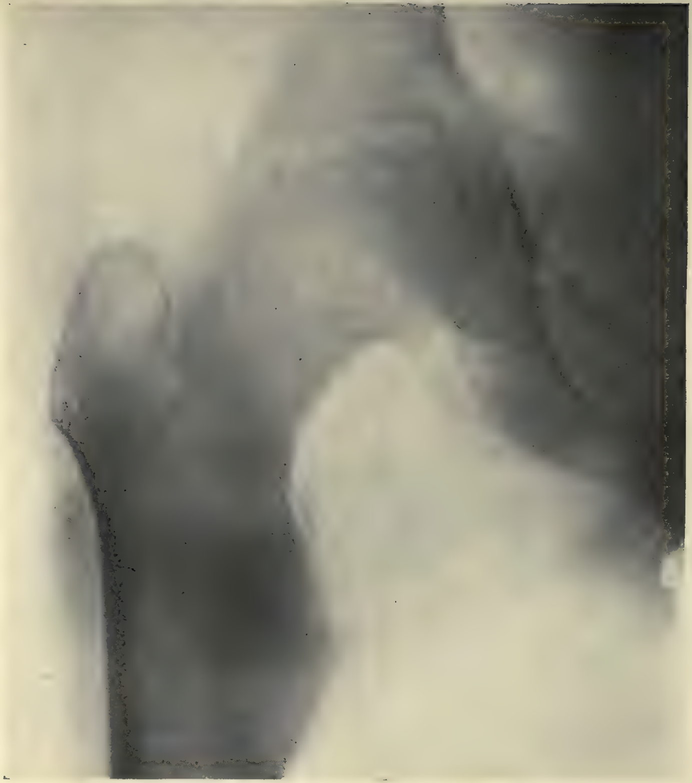

Fig. 225.—Skiagram made before operation. The ankylosis is seen to be not complete. The upper half of the joint cavity is visible, and the lower half is obliterated by the bony ankylosis which was present. The leg was slightly adducted and flexed at the hip.

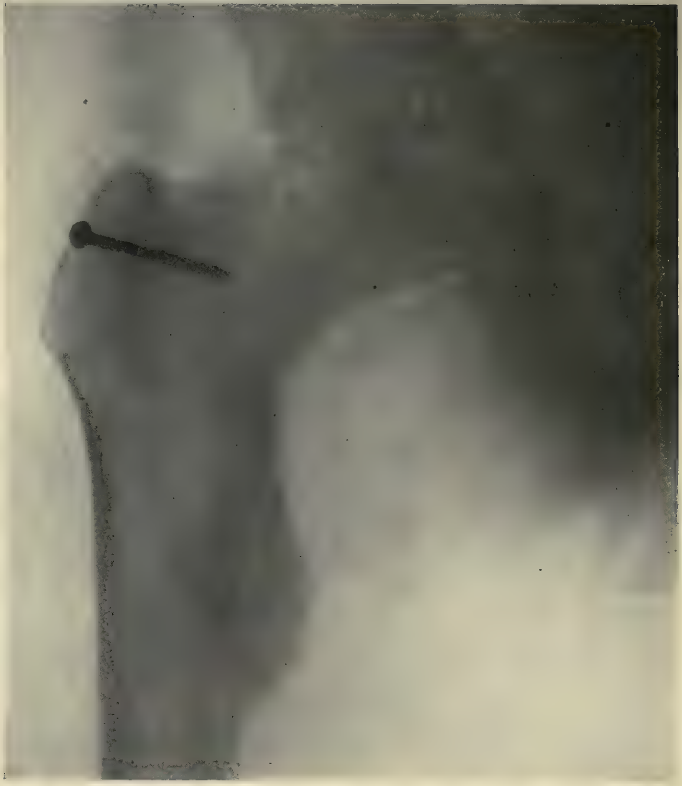

Fig. 226.—Skiagram made about five weeks after operation, when patient was up on crutches with good voluntary motion in the joint. The picture is not a very good one. The outlines of the joint cavity are not clear.

volved. There is also some absorption of the upper margin of the head with a fragmenting of the bone at that point. It looks as though he had a deposit of new bone around the acetabulum, because the shadow shows compact bony tissue. We are going to try to do an arthroplasty. The hip gives the best result after this operation. Fortunately, I did my *first* arthroplasty on the *hip-joint*—that was in 1902—with a perfect result. That is what encouraged me to go on with the work in ankylosis of other joints. The technic of an arthroplasty on the hip is much more simple or, better, less complicated than in the case of any other joint. The operation is easier to perform, the results are better and are secured sooner, with the possible exception of the mandible.

In the prophylactic treatment of these cases, if you are going to follow out our plan of treatment, *begin before* the joint is ankylosed; yes, of course, or it would not be a prophylactic treatment. *Yes, begin on the first or second day of the arthritis with the extension, and the* SAME *or a day later give the formalin in glycerin injection* INTO *the joint and maintain an* EFFICIENT *extension until the inflammatory process has fully subsided if you are going to call it the Murphy treatment.* If one deviates from these lines he should call it *his* treatment, and also his success or failure. It took nearly twenty years (see August CLINICS) to have our "early treatment of appendicitis" accepted. Let us hope the arthritides will not have to pay so frightful a toll before the treatment is accepted or disproved or supplanted. The injection of the 2 per cent. solution of formalin in glycerin and the Buck's extension have no value after the joint is destroyed. The treatment is preventive, not reparative. It must be done during the first days of the joint involvement to get the best results. It is the identical proposition of appendicitis all over again. We finally learned by clinical experience—not pathologic study—that general suppurative peritonitis, taken in time, was curable, but we have not learned to treat these infected joints in the same simple manner. It is not necessary to have a superlative degree of pathology before the joint is destroyed. After the joint is destroyed, it does no good to aspirate or inject or put on a Buck's extension—*do it at once!* Now is the accepted time for joint salvation.

In making my flap, I begin the incision about one inch below the anterosuperior iliac spine, sweep downward and backward for three inches and then upward, so as to encircle the trochanter, to a point 5 inches posterior to the starting-point. A straight downward incision 4 inches in length is then made from the lowermost point of the U-shaped incision. That gives us good access to the joint. The first incision extends down to the aponeurosis, including the fascia lata with the trochanteric bursa. Formerly we made a longer skin-flap than we are making now. It is unnecessary to do that. A short flap is equally as good. We get the interposing flap from the lower layers of this flap—the fat and deep fascia. We retract this flap upward. Formerly, we cut some of the anterior fibers of the gluteus medius muscle, but that is not necessary. Now I am ready to remove the trochanter with the muscles attached to it. I will pass the chain-saw underneath these muscles close to the neck of the bone, inside the trochanter, saw through the trochanter downward and outward, removing a portion of the muscle aponeurosis with it. By retracting that flap upward we have good access to the hip-joint. When we are ready to swing in the interposing flap, it will be carried down behind the head and neck of the bone, the inner portion of the flap covering the acetabulum so that the ankylosis cannot recur. The union of the femoral head with the ilium is not bony in the upper part, as far as we are able to estimate. It is fibrous. Only the lower part is a bony ankylosis. On moving the thigh the ilium and leg move as one mass. The ankylosis appears to be at the lower angle. That represents exactly what we saw in the skiagram. You will note that I am able to pass my scalpel between the femoral head and the acetabulum anywhere above, but not below. With a large curved carpenter's chisel we will cut through the ankylosis and free the femur from its attachment to the acetabulum. Now we have it free. There is a collar of bone around the head—new-formed bone—which we will remove. That collar of bone must all come off, otherwise we will have it impinging against the margin of the acetabulum and interfere with motion. This bone is all cicatricial. I must also take off the margin of the acetabulum. It is a little too high and will impinge against the side of the neck of the femur. This is cicatricial bone that I am removing from the margin of the acetabulum, just the same as the bone I removed from the neck. It is the result of the previous inflammations in the hip. Now we have the cavity fairly well prepared and the rough surfaces smoothed off. Next we will shape the head of the femur with the end mill, and ream out the acetabulum with the reamer. That smooths off the surface within. We have restored the normal conformation of the head. We have a perfect anatomic acetabulum again and a perfect head.

We are ready to place the interposing flap. We dissect off the fat and fascia from the original skin-flap, making it about ¼ to ½ inch thick. We drop it down behind the neck of the femur so that it will cover the whole head after it is replaced in the acetabulum. I will suture that to the shreds of the capsule on the neck below, so that it cannot get away. All raw bony surfaces are covered by this fat and fascia flap. I do not need a fixation on the top of the neck because the base of the flap fixes the top. All I need is fixation on the lower side of the neck. A few tacking sutures are also placed on the flap at the lower and anterior edges of the acetabulum rim. Now to replace the head in the acetabulum. We first make traction on the leg then internal rotation and abduction—and the reposition is completed. That practically completes the operation. Note how free the range of motion is in this joint now. There is no pressure on the flap. By putting an extension on this leg we will overcome muscular contraction after the operation and prevent a pressure necrosis of this flap. Next we replace the trochanter, securing it in position with a nail. That throws the muscles attached to the trochanter right back again in normal position so that the head cannot get out. We use an ordinary six-penny wire nail, driving it through the trochanter into the bone. More recently I am having these nails silvered because they are less likely to produce a softening of the bone around the nail. We rarely take these nails out. If they are properly put in, you do not have to take them out. You can see that that work is not as difficult as the work on the knee—not at all. It is much less difficult because less reconstruction needs to be done.

We have not taken off more than 1/3 inch of the head of the femur, so that we will have practically no shortening of the leg. We suture the cut edges of the muscle aponeurosis with chromicized catgut, so as to aid in securing fixation. That operation is in all its details practically identical with the first hip arthroplasty I did in 1902. I did it on the cadaver repeatedly before finally doing it on the patient. We close the skin incision with a few silkworm-gut sutures and horsehair. These sutures must not be tied tight enough to necrotize the tissues. No drain. The usual alcohol and phenol gauze dressing will be applied. Both legs will be dressed in an abducted position in a travois splint with a Buck extension of 20 pounds on the operated leg to keep the pressure of the contracting muscles from necrotizing the interposing flap. He cannot get an ankylosis again because there will be no bony contacting.

Let the record show that a U-shaped incision was made; that the trochanter with its muscular attachments was freed; that there was a bony ankylosis between the head and the acetabulum; that this was chiseled free, and a new head and a new acetabulum were made; that a flap from the deep fascia was dissected free and placed over the head of the femur and fastened to the acetabulum and around the neck of the femur; that the head was put into the new acetabulum; that the trochanter with its muscular attachments was nailed back into position with one nail; and that the wound was closed with horsehair.

[The patient got on splendidly. The stitches were removed on the fifteenth day. Primary wound healing. The patient was up on crutches in five weeks with good motion in the hip. He left the hospital July 2d with a normal range of practically painless motion in the hip.—Ed.]

CALCULUS IN THE URINARY BLADDER.—SUPRAPUBIC LITHOTOMY

HISTORY

PATIENT, male, aged sixteen, enters hospital on account of frequent and painful urination and pain in the bladder.

Fourteen months ago he began to have pain in the bladder, especially preceding urination, and he noticed that urination had increased in frequency. Since that time the pain has increased in degree and period of duration. He has some pain behind the symphysis all the time, and also in the perineum. The pain on urination is now very severe, and he often has difficulty in starting the flow of urine. Often the stream is interrupted for one or two minutes, and then it continues again. Occasionally he has very severe pain after urination. Ten months ago (July, 1912) he noticed blood in the urine for the first time. Since then he has passed blood six or seven times. Occasionally the blood was mixed with the urine, and at other times he passed bright red blood at the end of urination. He has also passed a stringy mucus. At the beginning of the trouble he had to get up only once at night to urinate, but now he urinates 7 to 15 times every night. Past history negative.

The skiagram in his case shows the presence of a stone of about the size of a hen's egg in the bladder.

COMMENTS AND OPERATION

DR. MURPHY (Operation May 3, 1913): Vesical calculi may be movable or fixed in diverticula. A stone may be held spasmodically in the upper part of the bladder, or it may be wedged behind an enlarged prostate. Multiple stones may be present. The symptoms in this case are typical of vesical calculus, and the skiagram shows the stone. For stone in the bladder we do the suprapubic operation—never anything else. Why do we prefer that operation to the perineal operation? If you do the perineal operation you disturb all the structures at the neck of the bladder, which is the urinary control zone, while if you do the suprapubic operation you just cut through the wall of the bladder and remove the stone. The interior of the bladder is easily accessible and can be explored thoroughly for stones, diverticula, etc.

First we pass a urethral sound into the bladder. The tip will serve as a guide later. Fixation of the sound is secured by putting forceps on the handle; that holds it squarely in place. You get your location and know just where the bladder is. The patient is placed in the Trendelenburg position. We make a three-inch incision in the median line from the symphysis pubis upward. I cut the attachment of the rectus on either side so as to let the muscle retract and give us freer access to the space of Retzius. The bladder is easily recognized by its muscular structure and its pink color. The tip of the sound is right in the field. I push the peritoneum back so that I will have plenty of room. By pressing the handle of the sound downward, the tip projects up into the field. Next we pass two silk sutures through the bladder-wall, one on either side of the tip of the sound. These sutures serve as anchoring and traction sutures. The ends are long and are secured with hemostats. We always use them in this operation. The bladder cannot now be displaced nor its margin lost sight of. You will note that there was practically no prevesical fat here. The bladder was exposed quickly. Next we divide the bladder between these anchor stitches, and we dilate the opening enough to remove the stone. As the urine pours out, it is immediately swabbed up with gauze sponges. We insert two fingers into the bladder and easily withdraw the calculus. It is smooth, oval in shape, and about the size of a hen's egg. Then we explore the inside of the bladder with the finger, to make sure that there is nothing else there. There is not. The urethral sound is now withdrawn. We suture this wound in the bladder-wall with fine catgut, not including the mucosa, put a rubber tissue drain into the space of Retzius, and close the ab-

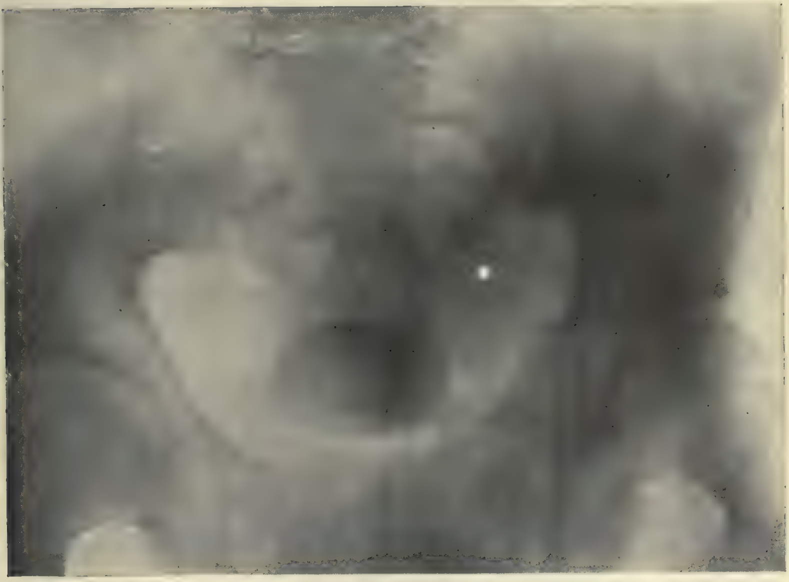

Fig. 227.—Calculus in the bladder. A stone about the size of a hen's egg is plainly visible in the pelvic basin in the skiagram.

dominal wall in the usual way. We first withdraw the anchor stitches and fix the drain to the abdominal wall with a silkworm-gut fixation suture. Next we suture the rectus muscle and its aponeurosis, and then we close the skin incision with silkworm-gut tension sutures and horsehair.

Let the record show that when the sound was inserted into the bladder one could hear a click as though striking a stone; that a median incision was made over the space of Retzius; that the anchor stitches were put into the bladder and the bladder opened; that a stone the size of a hen's egg was removed and the bladder closed; that a small gutta-percha drain was inserted in the space of Retzius, but no drain was put into the vesical cavity; and that no catheter was put into the urethra.

Let the record further show that Sister reports the sponges counted and correct.

[The patient made a rapid and smooth recovery. Stitches removed on twelfth day. He left the hospital on the twenty-fourth day.—Ed.]

TUMOR OF THE TONGUE—TUBERCULOMA

HISTORY

Patient, female, single, aged twenty-one, enters hospital on account of mass near the midline of the tongue, an inch and a half back from the tip, and apparently about the size of an almond. She first noticed this mass about six months ago. At that time it was hard and indurated, and of about the size of a pea. It has gradually increased until at present it is the size of an almond, hard, regular, with a slimy surface. It has never ulcerated nor bled. She has never had a chill or fever. There is no glandular enlargement. She is free from continuous pain, but has discomfort in talking and eating, and hard substances irritate the tumor and make it severely painful. She has no chronic cough or night-sweats or evening rise of temperature. For the past twelve years she has had occasional eruptions on the legs and arms in the form of pimples. These eruptions persist for ten days or two weeks and then disappear. They never form pustules unless they are scratched, but she says that, on healing, they leave brownish spots. She has had no chronic sore throat or falling out of the hair. Menstrual history negative. Has had leukorrhea for past four or five years. Had tonsils removed two years ago. Family history negative to tuberculosis and carcinoma.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 24th): This is one of the cases in which we have resorted to all kinds of tests and yet we have not arrived at a diagnosis. To aid us in the differential diagnosis I have prepared an outline of tumors of the tongue:

TUMORS OF THE TONGUE

A. Benign—epithelial { Thyroglossal duct derivatives. Adenomata.
B. Benign—connective tissue.
  Fibroma; neurofibroma; lipoma; myxoma; myoma (macroglossus muscularis); chondroma; osteoma.
C. Benign—vascular.
  Hemangioma.
    Cavernous angioma.
    Cirsoid angioma.
    Lymphangioma (macroglossus)
    Lymphangioma cysticus.
D. Embryonal defects.
  Teratoma.
  Dermoid.
  Cysts.
E. Malignant tumors.
  Sarcoma.
  Carcinoma.
F. Infective granulomata.
  Tuberculoma.
  Syphiloma.
  Actinomycosis.
  Glanders.
  Leprosy.

The most common types of non-malignant tumors of the tongue are—First: Congenital tumors—tumors that take their origin in remnants of the thyroglossal duct. Second: The next class, and one very common, includes the tumors in the vascular structures that occur at the base of the tongue. We divide them into three classes: (1) The lymphangiomata, that occur in the lymph-spaces. They often reach an enormous size (macroglossus), so that the tongue protrudes from the mouth. (2) The lymphangioma cysticus—less common. The soft, pliable, compressible, non-malignant tumor, diagnosed often as a sarcoma. (3) The hemangic vascular tumors. Capillary neoplasms of the tongue are very rare. We find there cysts of the vessels occurring in the mesoblastic elements of the tongue. They produce all the symptoms that the mesoblastic tumors produce in other portions of the body. There is only one malignant mesoblastic tumor of the tongue, namely, sarcoma. These are extremely fatal. They are fatal in the fact that they grow rapidly and that they metastasize early.

Other non-malignant conditions that occur there are also the infectious granulomata, syphiloma, tuberculoma, leprosy, glan-

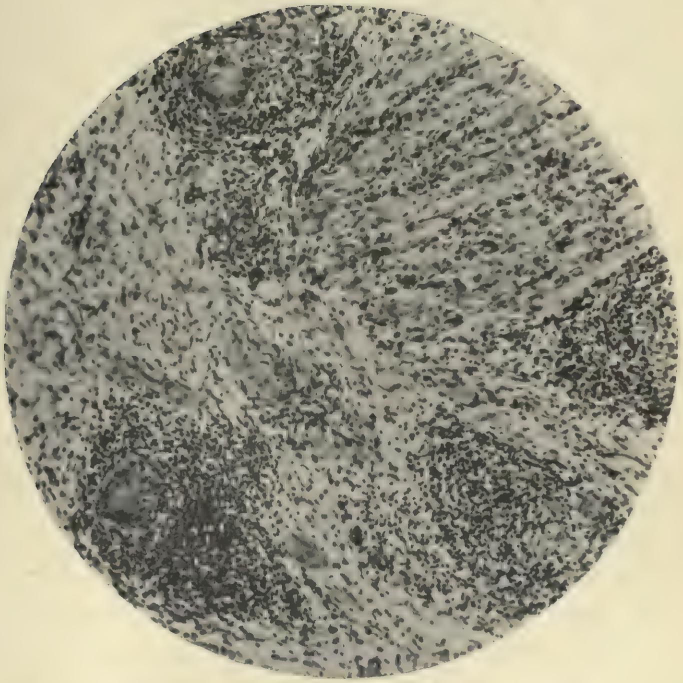

Fig. 228.—Tuberculosis of the tongue. Microscopic section, showing portions of four miliary tubercles, with giant-cells in two of them. There is also a general infiltration of the tissues by epithelioid new-formed round-cells.

ders, and actinomycosis. These tumors result from infections of the tongue. Almost all of them come from a primary lesion in the mucosa or submucosa.

Here we have a tumor that I have not been able to classify. The Wassermann test and the tuberculin test have been made in this case and both have been negative. It is one of the most peculiar cases that has come under my observation. It is near the midline, and goes down well into the base of the tongue. It seems to be situated in the muscularis of the tongue. We will not take out that tumor until I have removed a portion of it for microscopic examination by two or three pathologists. If an operation is finally indicated, it will have to be a very radical one. The operation today is done merely to secure a specimen for microscopic examination. She gives no history of trauma or syphilis. I will remove a large portion of the center of the tumor and close the gap with catgut sutures. Small fragments of tissue should not be relied on for microscopic examination.

(After the microscopic examination.)

Regarding the tumor of the tongue which we had a few days ago (mentioned above), I would say that the microscopists pronounced it a tuberculoma. It looked like a sarcoma clinically, but we would not operate on it as a sarcoma until we had decided definitely that it was such a tumor. Remember, there was no ulceration on the surface, which is so typical of tuberculosis, and that there was a deep infiltration of the tongue. (She was given the Wassermann and tuberculin tests and both were negative.) It was a solid tumor. After going over all the tests we concluded that we could not make a differential diagnosis. The microscopists found, on the surface of the mass sent them, some typical miliary tubercles. That settles the diagnosis. She will not be operated on, but will be given tuberculin injections.

About four weeks ago we saw another case of tuberculosis of the tongue with ulcers on both sides. It looked very much like a carcinoma, but there were areas of repair. The patient was a male, fifty-three years of age. The first lesion appeared two years before we saw him. When he came to us he had numerous ulcers all over the tongue. It was enormously swollen, so that he could not talk. We advised that man to take the tuberculin treatment. We gave him 5 minims of the Lucius and Brüning tuberculin dilution No. 3 every fifth day for five weeks, when he refused further treatment and left the hospital. In making the differential diagnosis between tuberculosis and carcinoma, remember you are very much aided by these areas of repair. In carcinoma, unless it has been cauterized or treated by the Röntgen ray, there is no repair of tissue; the destructive process is a continuous one, while in the tubercular process repair commonly goes on in one area while destruction of tissue is taking place in another. If you find in one place on the surface an ulcer that is healing, and in another one an area that is breaking down, you can conclude at once that it is not a carcinoma. That is a very valuable point to remember in connection with these cases, but note the danger of error: if the malignant mass has been cauterized or cut into, it may heal in one place and break down in another.

In the case of the tongue tuberculosis just cited, where the tubercles were on the surface and ulcerated, we had a very pronounced tuberculin reaction, because there was a mixed infection. I am commencing to believe that a mixed infection is essential in the production of a high temperature tuberculin reaction. In this case there was a tuberculosis present. It did not look like a carcinoma. The other case was different. It was a tuberculoma. It might have been an adenoma. Adenomata of the tongue are rather rare, but they do occur, usually in the sublingual glands, rarely in the muscularis of the tongue. I think we showed good judgment in examining the section before doing a complete operation. It would have been awful to have taken out that girl's tongue. Now, we have to treat her for her tuberculosis with tuberculin, and we will probably also treat her with the Röntgen ray. We believe, however, that we can cure her with the tuberculin alone because it is a tuberculoma. The process of repair will be slow, because that is the rule when muscle tissue is the seat of the tuberculosis.

[The patient received one injection of tuberculin and then returned home with instructions to continue the treatment.—Ed.]

The following is a most interesting case of carcinoma of the tongue on account of the youth of the patient and long presence of the disease, which was probably not originally malignant, but is now unquestionably malignant. The condition of the patient is hopeless. Surgery offers no relief. Procrastination is responsible for a great percentage of cancer mortality.

CARCINOMA OF THE TONGUE.—PATIENT BROUGHT IN FOR EXAMINATION—SPECIMEN REMOVED FOR LABORATORY EXAMINATION

HISTORY

Patient, male, aged thirty-one, enters hospital complaining of soreness on the right side of the tongue. The tongue on the right side is hard and rigid, and there is present a discharging abscess. The trouble began five or six years ago, with soreness on the right side of the tongue. His dentist says that there was no rough or jagged tooth preceding this, but that he always had a slight speech impediment, which seemed to be inherent in the tongue. He consulted a physician when the trouble first began, and was told that it was nothing serious. The condition remained stationary for about a year, when he first noticed a discoloration on the right side of the tongue. There was no further change for some time. The tongue remained a little sore, tender, and discolored until the spring of 1912, when he again consulted a physician, who applied trichloracetic acid to the indurated area. A slough formed and became separated from the tongue. This did not seem to check the progress of the lesion; in fact, it seemed to be gradually increasing in extent. October, 1912, he consulted a physical culture exponent who told him that he had tuberculosis of the throat, and he has taken treatment in the form of exercise from this man ever since. In the past three months he has lost 12 pounds in weight, and about three weeks ago he noticed a swelling under the mandible on the right side. He says he had a similar mass at the angle of the jaw several months ago, but it has disappeared. During the past year the lesion has been abraded,—like an ulcer,—discharges a little pus, and occasionally cheesy-looking particles. About four months ago he began having a constant dull pain around and in the right ear. This pain troubles him less now, and it has not been constant during the past three weeks.

Past history is negative. He rarely indulges in alcoholics; smokes one cigar a day. He has not been around horses or cattle, and is not in the habit of chewing straws, exposing him to actinomycosis. Gonorrhea five and one-half years ago. Says Wassermann is negative. Family history shows no tuberculosis or carcinoma. Mother, five sisters, and one brother living and well. Father died of apoplexy at sixty-eight.

Examination by Dr. Murphy (June 20, 1913): The right side of the tongue is hard and woody, back as far as I can reach, including the whole base of the tongue. There is a sinus leading down to a tooth and this is discharging pus slightly. Enlarged gland under right mandible.

COMMENTS AND OPERATION

Dr. Murphy: There, you see, is a thickened tongue. It is ulcerated. The whole base of the tongue, all the way across, is infiltrated. We think here of one of four lesions—tuberculosis, carcinoma, syphilis, and actinomycosis. Why should he have this pus discharge? It is not very great, but just enough to show that there is an infection in his tongue. The test we are going to give him today is the tuberculin test; that is, 5 minims of dilution No. 2 of Lucius and Brüning's Koch's old tuberculin, injected hypodermically. The temperature will be taken every two hours regularly. If it is an active tuberculosis, he will get an elevation of temperature in from twenty to twenty-eight hours, with chill, malaise, possible headache, and nausea. If it is not an active tuberculosis, the reaction will not be severe. We will have a Wassermann test made to see if it is a specific lesion.

This is a beautiful case. First he had a roughening on the side of the tongue. He consulted a dentist, who did some beautiful dental work, cleaned the teeth, and filled the cavities. He had been under treatment about a year. Then he went to a physician who applied trichloracetic acid, which did not relieve the trouble. Then he consulted a physical culture expert (he did not call himself an osteopath), and he has been giving the patient exercises and rubbing this cheek ever since. A few weeks ago he noticed a swelling in his neck under the jaw-bone. The physical cure man rubbed this gland, which, of course, was only a late symptom of the actual trouble. Three-fourths of that man's tongue is infiltrated with a neoplasm. If it is an actinomycosis, we will probably be able to save his tongue. If it is a tuberculosis, we hope to save the tongue. If it is a gumma, we will save his tongue. If it is a carcinoma—and I think it is—we could take out the tongue, although the disease has progressed so far that after further consideration we will discourage surgical treatment. He came here originally for treatment of "neuralgia" in his ear, and incidentally he showed me the tongue.

You may divide the tumors of the tongue into, first, the malignant group; second, the granuloma group; and third, the congenital group. Of the malignant group, you have two types of tumors in the tongue—one, the superficial ones, that occur in the stratified squamous epithelium, a carcinoma of the tongue, usually initiated by some type of irritation of the surface slowly penetrating from the surface downward and involving a greater or lesser portion of the tongue, depending on how soon it is interfered with by the surgeon. Second, sarcoma of the tongue. Sarcoma of the tongue is rare. It does occur, but most often in the intermuscular tissue or in the connective tissue of the tongue. Third, tumors that occur from embryonal defects of the thyroglossal duct, which you know opens at the apex of the V formed by the circumvallate papillæ, and extends downward to the thyroid. It usually closes before birth, its location being marked by a dimple on the tongue. It may not be obliterated entirely, however, and then it becomes the seat of malignant disease—carcinoma or sarcoma. As to the granulomata, the Wassermann test in this case was negative; the tuberculin test was negative, ruling out syphilis and tuberculosis. Next is actinomycosis. There is nothing in the history of this case to point to actinomycosis; but a microscopic examination alone will make the diagnosis. The enlarged lymph-nodes under the jaw-bone, however, may point to any of the granulomata that occur in the tongue. Tuberculosis commonly appears as a surface ulcer, and occasionally penetrates "taproot"-like down into the tongue. You will remember we had one case, a typical tuberculosis of the tongue, that very closely resembled a sarcoma (p. 871). It occurred in a young woman. This patient is thirty-one; the other patient was twenty-one. We took out a section for microscopic examination, and it was a most beautiful specimen of tuberculosis of the tongue. That patient is being treated by injections of tuberculin. Remember, the tongue is a muscular structure which yields slowly to the action of tuberculin. It repairs slowly, but we hope to secure repair without extirpation. If this patient today has tuberculosis, then it may be cured by hypodermic injections of tuberculin. If not, then we may have to amputate his tongue way down to the base, which is a serious procedure.

This lesion has been present a long time. We know he has had a disturbance in his tongue for four or five years. It is possible, notwithstanding his youth, that this is a carcinoma of the tongue; that it was a papilloma originally, which finally became cancerous. It does not look like a cancer. The clinical history is not altogether that of cancer. It does not feel like a cancer. Considering the size of the lesion, an operation for cancer would not do much good. If it is a carcinoma, I would not advise a tongue excision, because there is bound to be a recurrence, and his life would not be materially prolonged. I feel that he will live longer and better if no operation is done—no attempt made to remove his tongue. We are going to take out enough tissue today to make a microscopic examination. It is such a gross, grave lesion that it frightens us to touch it. This is a serious situation. When I press on that mass in the tongue there is a material oozing out, the type of material that you find in actinomycosis—a sanious fluid containing little yellow granules—the so-called sulphur granules. They look like actinomyces. There is little hemorrhage as we incise the tongue, meaning that the infiltration is so severe that there is no chance for hemorrhage. The blood-vessels are compressed or occluded.

(Examining specimen removed.) Here is a spot that looks exactly like an actinomycotic lesion—a little yellow mass. Still there are other types of mycoses, and we cannot base a diagnosis on that little yellow granule alone.

Let the record show that a portion of the tongue was removed and sent to the laboratory for diagnosis.

Laboratory Diagnosis.—From many slides—unquestionably squamous-cell carcinoma. [The patient was advised to have radium treatments and Röntgen-ray exposures. Here is a case where the gross appearance and the clinical history were in favor of a granuloma and the microscope showed it an unquestioned carcinoma. In our work when the clinical history and gross appearance do not correspond with the microscopic findings, we always insist on many verifications of the latter.—Ed.]

TUMOR OF FEMUR; CAVITY FILLED WITH MOORHOF WAX

HISTORY

Patient, male, aged twenty-six, enters hospital on account of pain and tenderness in the right leg, midway between the knee and the hip. For the past sixteen months (since January, 1912), he has had shooting pains along the back of the right leg down to the foot. He says the trouble began after a long drive on a very cold day, when his legs got very cold. He had slight pain last December on the outer side of the leg at the site of the present tenderness. He now has a constant aching pain, with acute exacerbations, the pain extending down the outer side of the leg to the ankle. These pains are worse at night and when leg is fully flexed. Walking does not cause pain. At present the leg seems to be larger than its fellow in the middle of the thigh and it is very tender. There is a mass to be felt that appears to be a part of the bone, but it is not as hard. He does not remember sustaining an injury at this point. The pain was not preceded by a chill, a cold, a sore throat, or a cough. Past and family history negative. Venereal infection denied.

He was examined two weeks ago and was told that it was a sarcoma of the femur. When he was examined last December for the pain the mass was not detected.

COMMENTS AND OPERATION

Dr. Murphy (May 10, 1913): When did he drive in the cold?

Intern: January, 1912.

Dr. Murphy: Was that the January following the December in which he had the pain?

Intern: He had the pain in December, 1912, eleven months later.

Dr. Murphy: When did he have his Neisserian infection?

INTERN: He denies any Neisserian infection.

DR. MURPHY: Did you have the urine examined?

INTERN: No.

DR. MURPHY: The urine should have been examined to see whether it contained the so-called "Tripper-faden." After a Neisserian infection these threads may be found in the urine for the remainder of life in an enormous majority of the cases, and always in the cases in which there is a metastatic infection. That is very valuable from a practical standpoint. You can see these threads every time if you take the trouble to look for them.

The typhoid and the Neisserian metastatic infections are two most painful bone infections. They are the two conditions that should be eliminated always in making a differential diagnosis. This man denies having had both typhoid and gonorrhea. The question that comes up is, did the trouble commence in December, 1912, with the pain, or did it originate the day following the exposure and only manifest itself symptomatically in December? If the latter, it is an infection tumor; if the former, it may be a neoplasm. The skiagram shows that the medulla of the bone is clean cut. Careful study shows that there is a hyperplastic ossification; not the absorption and ossification that come with a sarcoma, but a hyperplastic ossification that occurs in injuries and as the result of long-continued infection irritation.

We have placed on the board this morning a classification of tumors of bone:

BONE TUMORS

Osteoma eburnum
    (compact osteomata).
Osteoma spongiosum.
Osteoma medullare.
Parasitic bone tumors.
    A. Cysticercus cellulosæ.
    B. Echinococcus cyst.
Granulomata.
    Tuberculosis.
    Actinomycosis.
    Syphilis (gumma).

Infective:
  Gonococcus (abscess).
  Typhoid (abscess).
  Staphylococcus (abscess).
Fibroma; osteitis fibrosa cysticus.
Chondroma.
Chordoma.
Chloroma.
Myeloma.
Thyroid (benign; malignant; both metastatic).
Carcinoma (secondary).
Sarcoma.

First, there is the eburnating tumor, a variety of which we know practically nothing. Second, the spongiosum tumor, a variety of which we know almost nothing, although now some experiments are being carried on in the General Hospital in Rome to determine its nature. Third, the medullary tumors are beginning to be considered much more frequently the result of some type of infection. The most common of the medullary tumors is the class known as infective tumors. Fourth, the type of neoplasms known as osteofibroma or osteitis fibrosa cysticus. It is the most common of the non-malignant lesions of bone. It begins in the shaft of the long bones, usually in the metaphysis, never in the epiphysis. The skiagram is as important in making the differential diagnosis as is the microscopic section, because it nearly always differentiates the sarcoma from the osteitis fibrosa cysticus, when it has developed to any considerable size. The osteitis fibrosa cysticus forms pockets of varying size and number. The bone enlarges uniformly, as a fusiform swelling, and the tumor is honeycombed with pockets. There is no invasion of the surrounding tissues. The periosteum may be slightly thickened, but otherwise it is unchanged. A sarcoma never does that. It gives just the opposite picture. It is irregular in contour and density; is apt to pulsate; causes distention of the superficial veins, and may give a crackling sensation on pressure, owing to thinning of the bone. The skiagram may show a sharply limited shadow, but rarely a uniform enlargement. Metastases may form. The diagnosis may be very difficult and often it can be made only after an exploratory incision.

The chondromata are cartilaginous tumors. The chloroma is the type that occurs mostly in the skull. On incision the tumor is seen to have a beautiful green color. It is recognizable at once by its color. The myeloma is a non-malignant lesion occurring usually in the epiphysis of bones, rarely ever in the shaft of long bones. The myeloma, of which we had two cases recently, resembles a sarcoma in its gross appearances. In our cases when the tissue was examined by the microscopist it proved to be non-malignant. In one case the cavity in the tumor was filled with Beck's paste. What happened? The patient was not benefited at all. The paste acted as an irritant and the condition became steadily worse. Beck does not advise the use of his paste for relief of this condition. If they had filled the cavity with a blood-clot or with the broken-down framework of the canopy and sides of the cavity, as we did when we operated, the trouble would have been cured. We chiseled off the canopy and sides, and dropped the bone fragments into the cavity. The wound healed by primary union. The cavity will be filled with compact bony tissue, because the bone fragments play the same rôle as a bone transplant.

The next lesion to be considered is the sarcoma. If it is the vascular type of sarcoma, we do not have this rarefied picture. We should have a dense picture rather than a light one. Does that type fit this case? There is no history to that effect.

As to the diagnosis of the type of tumor from macroscopic and microscopic examination: What are the gross and what are the microscopic interpretations of the tumor? It sounds rather unscientific to say that where the gross specimen is that of benignity the clinical history of the case is apparently that of malignancy. We rely to a very great extent on the clinical history and the gross appearance of the tumor; in fact, we give these the preference over microscopic sections. For a long time we believed that the microscopist erred in interpreting certain

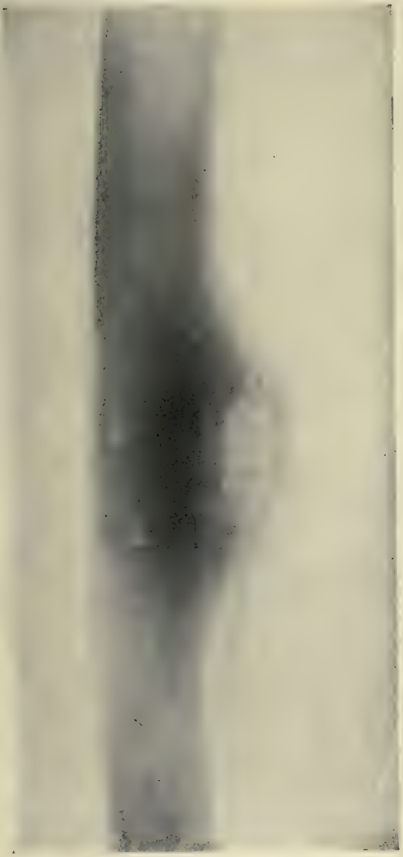

Fig. 229.—Anteroposterior view of a case of tumor of the femur. The bony mass to the right of the femur is somewhat irregular in outline and indistinct. It is a subperiosteal growth.

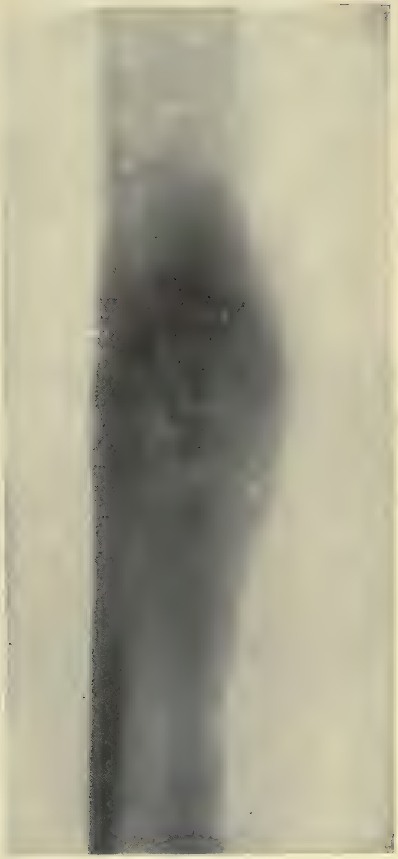

Fig. 230.—Lateral view of same case. The tumor appears to be dense. This is due to the fact that the ray passed through the entire thickness of the femur.

tumors of the embryoplastic type, as, namely, of the benign type of sarcoma in bone, as being non-malignant, when the clinical history showed these tumors to be malignant. This shows that the microscope cannot be relied on solely in estimating the *malignancy;* that three or four men may agree in the interpretation of a microscopic specimen that it is malignant—and still it is benign clinically; malignant microscopically and benign clinically. Fortunately, the cases of that class are comparatively few in number, but we must remember that they do occur. We may have to regret it when we do a radical operation in a case of tumor and find that extensive secondary microscopic examination shows it to be probably non-malignant. And we have to regret it on the other side, and I think more frequently, when a case is interpreted by us clinically as being non-malignant, also by the microscopist as being non-malignant, and we fail to do a radical operation and the clinical course proves that it was malignant by its recurrence, terminating fatally. We may err both ways.

If you will look at the skiagram again you will note that the highest point of the tumor is just beneath the profundus artery. The structures of the thigh get their arterial blood-supply from the femoral artery, the direct continuation of the external iliac. The popliteal artery is the continuation of the femoral and divides into the anterior and posterior tibial below the knee. The profunda femoris or deep femoral lies so close to this mass that we must bear it in mind all the time during the operation.

We will place a constrictor on this leg. I am going to make my incision between the sartorius and the vastus internus, avoiding the femoral vessels and the anterior crural nerve. We displace the muscles inward. The deep femoral rests right on the periosteum and has three main branches and four perforating branches. It supplies most of the muscles in this region. There are so many important vessels here that we must be careful in using the instruments.

When I flex the thigh slightly, and likewise the knee, I am able to relax all this internal group of muscles which makes up the internal hamstring. I have the vessels all displaced inward.

I thought I could get at that tumor from behind, but I find now that it comes forward instead of backward. I think I will do better by going in in front.

There is the femoral artery. The sartorius lies very close to it. There are the fibers of the anterior crural nerve, which run along in that sheath with the artery just to the outer side. By retracting all these muscles and vessels inward I am onto the tumor. It is on the inner aspect of the femur. There is the deep profundus right above the tumor. I will take out a section from the anterior surface of the tumor with a small straight chisel about ¼ inch wide. That feels very much as though I was cutting hard, compact bone.

There is a pocket of granulation tissue, and another. It looks like a central granuloma with compact bony tissue surrounding it. That means that this is a type of infection: that it is not a sarcoma, which is exactly in consonance with the skiagram. That is what caused the irritation. That is mighty gratifying—gratifying to us and gratifying to the patient, because we do not have to do an amputation or an excision of the shaft of the bone. That is my interpretation of the gross specimen. We will have this tissue examined microscopically.

We will clean out all this cavity with a sharp curet. It resembles in its gross appearances a divided granuloma. There is a small sequestrum in the center of the cavity. It does not look like a tuberculoma. I interpret this as a granuloma and not as a sarcoma. If it is a granuloma, it is very important that it should not become infected. What does that hard, compact bone mean? It is a cicatricial bony tissue, just like cicatricial tissue that surrounds an abscess in the softer structures. Cicatricial bone is compact bone. It is caused by irritation. After we have cleaned out all this granulation tissue we will fill this cavity with Moorhof's bone wax, which consists of iodoform, 20 parts; spermaceti, 40 parts, and oil of sesame, 40 parts. The cavity must be dry and sterile before the wax, heated to 50° C., is poured into the cavity. You know what the dentist does. We want to do what the dentist does—that is, dry-fill the cavity. That mixture will solidify. The wax is ultimately absorbed and replaced by fibrous tissue or bone. In five or six minutes the mass will be hard enough; then I will take off the constrictor. There will not be much bleeding, because we displaced the vessels to one side.

Let the record show that the deep profunda was right at the tumor; that there was a hard, bony mass, thicker and harder than normal bone; that there was a pocket of granulation tissue at its apex; that a window was cut into the mass and a piece removed for microscopic examination; that the cavity was cureted and then filled with a Moorhof plug.

The wound is closed with horsehair, and an alcohol and phenol gauze dressing is applied.

Laboratory Report.—Mixed-cell sarcoma, containing large round and large spindle-cells with lime deposits.

[The pathologist's diagnosis of the tissue did not confirm the clinical diagnosis of granuloma. The history and skiagram confirmed the clinical diagnosis. The patient left the hospital within three weeks feeling free from pain and discomfort. The subsequent course of this case may determine which of these interpretations was correct.—Ed.]

ABDOMINAL FECAL FISTULA FOLLOWING PUNCTURE OF UTERUS BY CURET AND DRAINAGE OF RETRO-UTERINE ABSCESS. REMARKS ON USE OF CURET.—RESECTION OF BOWEL.—END-TO-SIDE SUTURE. —ANASTOMOSIS

HISTORY

PATIENT, female, aged twenty-seven, married, enters hospital because of a fecal fistula, pain in abdomen, extreme emaciation, and weakness. February 6, 1912, she was cureted because of pain in the lower abdomen and cessation of menstrual flow. She had been nursing a baby sixteen months old, and had not menstruated since last pregnancy. During the curetment the uterus was punctured and a loop of bowel was pulled down through the uterus and appeared at the vulva. It was pushed back. February 10, 1912, she entered a hospital suffering with peritonitis. February 22, 1912, the abdomen was opened and a large abscess found back of the uterus. This was drained through the old puncture in the uterus out through the vagina. The next day fecal matter was discharged from the abdominal wound and from the vagina. The following day and thereafter the fecal discharge was through the abdominal wound only. She has always had some normal bowel movements. April 10, 1912, she left the hospital. The fecal fistula was discharging very little. It was almost closed. Three weeks later, on attempting to walk for the first time, she had a severe chill and temperature went up to 104° F., with morning remissions. May 15, 1912, the abdomen was opened again and another abscess was found behind the uterus and drained through the abdominal wound. Fistula then discharged pus for a few days and then large amounts of semisolid fecal matter. Most of the feces is still discharged through the fistula, but patient has a slight bowel movement daily.

COMMENTS AND OPERATION

Dr. Murphy (Operation June 24, 1913): Where was the curetment done?

Intern: At her home.

Dr. Murphy: At home! That is the point I have been making for the past seven years about the curet, namely, that the careless doctor carries his tooth-brush and curet in the same pocket and uses them both with about the same frequency. He pulls the patient over to the side of the bed, and with a sharp curet scrapes away to his heart's content. That is the way the curet has been used by many men for a long time. We do not do anything like that on surfaces that we can see; why do we curet the uterus with a sharp instrument just because it is hidden and we cannot see the mischief that is being done?

This is the third case that has come under our observation of a curetment with rupture of the uterus and descent of the intestine into the vagina. One of the earliest cases was one in which the uterus was cureted and punctured. The doctor did not recognize that he had punctured the uterus. Soon after the operation the patient had an elevation of temperature. The next day or the second day, when the doctor examined the vagina, he found a mass in it. For some reason he got the idea that it was the fetal cord, and he commenced pulling on it. It was the bowel, and he pulled off its mesentery. If he had thought of his anatomy, he would have known that the fetal cord has no mesentery. He brought 18 inches of the large intestine out into the vagina. Then he became frightened and brought the patient to the hospital. She had a temperature of 105° F. We cleaned out the vagina, cleaned off the intestine, and then did a laparotomy. We resected the 18 inches of large intestine, made an end-to-end union, and the patient recovered.

The next case was one of which I only heard the story over the 'phone. The doctor who called me said he had brought out something like 8 feet of the small intestine into the vagina through a hole in the uterus during a curetage, and asked me what he should do. I outlined the plan of operation. A surgeon operated on that plan and the patient got well.

The case before us today is another one where the uterus was cureted, punctured, and the intestine drawn out through the vagina. The doctor did not have the courage to call for assistance when he did not have the knowledge needed to treat the patient properly. He put back the intestine at once, pushed it back with the scissors, heedless of whether he got it back into the uterus or into the abdominal cavity. I cannot imagine a man being able to push the intestine into the abdominal cavity through the cervix of the uterus. The patient, however, developed a double fistula and an enormous suppuration in the pelvis and repeated abscesses. You can imagine how unpleasant it is to open this abdomen and disentangle these intestines.

The intestine that is involved in the fecal fistula in this case is the small intestine; at least we hope it is, because a small intestine fecal fistula is easier to handle than a large intestine fistula. You can resect the bowel any distance from the point of involvement in healthy tissue and make an end-to-end or end-to-side union and it heals. In the large intestine you can suture the fundus of uterus to the bowel and get a union that way, using the fundus of the uterus to fill up the gap in the intestine and save the patient, as I have done on two occasions when freeing of the bowel was impossible on account of the infiltration. It appears, from the examination, that in this case the small intestine is involved. We gave her an enema of three pints of methylene-blue and none of it came out through the wound. If this fistula were in the large intestine, the methylene-blue would have come out through the wound. Then we gave her some strawberry jam by mouth, and the seeds came out through the fistula. That is the best test for the location of a fecal fistula. If you will feed the patient strawberry jam for three or four days, the seeds will come out through the fistula if it is intestinal. The seeds will come out where gas and feces will not. Therefore, we concluded, from the methylene-blue not giving a positive response and from the seeds giving a positive response, that it was a small intestine fistula.

The diagnosis now having been made, you plan your line of attack. We are going to have a mass of adhesions to encounter. How many openings will I find in the small intestine? I have found as high as 20 with one cloaca or pocket. That means a hard time getting the adhesions freed. Where there are a number of openings in a small area we resect that portion of bowel and make an end-to-end union with the round button or with the suture. Sometimes we make a side-to-side union with the oblong button. The oblong button is very applicable to side-to-side unions, because it does not tend to produce a dilatation in the pocket above the button.

The danger of peritonitis from this type of fistula is not so great, because we are working in an infiltrated tissue with little free peritoneum in the field and the lymph-spaces are blocked. The chances of a non-union are increased by the absence of peritoneum, because here we have two connective-tissue surfaces that do not adhere as quickly as do serous surfaces, *i. e.*, intestine that is covered with normal peritoneum. There is still another possibility; locate the proximal coil of bowel where it enters the mass of adhesions and the distal coil where it exits from it; exclude that portion of bowel which is looped up into the mass of adhesions. Now, by making an end-to-end or end-to-side union you side-track the adherent coils. These may be removed later when the patient is in good condition to withstand such extensive procedure.

In attacking one of these cases you must be able to use all the resources available to get the patient out of the operation alive and into a position of possible cure. You have to consider all the possibilities in order to save the patient's life. If this intestine is still implanted in the uterus, you can see what a hard time I will have to free it. I will have to split the uterus and take the intestine out of the uterus. I cannot leave it there. I will have to split the uterus and dissect free whatever portion of bowel may be in it. No intestine is protruding from the cervix; nothing is to be felt, but that does not mean that the doctor got the intestine back into the abdomen, because she had a utero-intestinal fistula and a number of operations were done above, in an effort to open the abscesses which followed the curetment.

In the consideration of the question of curetment as a general proposition *no one should ever use a sharp, razor-blade curet.* Always use a dull curet—one with a flexible handle. A spoon that is stiff enough and sharp enough to take off the mucosa with its glands leaves nothing in that uterus when repair is complete but a mass of connective tissue which heals by a cicatrix formation without being covered over with epithelial cells. If, on the other hand, you use a dull curet and scrape off the surface only, the edematous mucosa, you leave the glands beneath, which furnish new epithelial cells to cover the abraded or denuded surface and make it possible for a normal mucosa to result. If you were to examine that uterus four weeks later, you would not know that it had ever been cureted, while if you were to examine the uterus in the other cases where a sharp curet was used, you would find a solid cicatricial mass, and no epithelial cells of the normal uterine type covering it. What happens to one of these young women who are cureted with a sharp curet? She is cureted usually because she has a menorrhagia or a painful menstruation because of a contraction of the os internum or the os externum. She always is cureted, whether she needs it or not. If you curet with a dull, flexible-handled spoon, you cannot possibly destroy the mucosa. You can scrape the mucosa until you are "black in the face" and you will not dig it out. If, on the other hand, you use that new sharp curet that has been on the market for the last few years, you take out all the mucosa and leave nothing but a muscular and connective-tissue lining. What happens to these young women? They are rendered sterile; they are rendered invalids because they do not have their normal menstruation. The menstrual period is cut down to a day or half a day, and then they commence to have all the neuroses that are associated with a premature menopause. For a long time it was believed that it was necessary to take out the ovaries to cause these neuroses, but you have the same type of premature menopause from a destruction of the mucosa that you have when you take out the ovaries. A curetment should be done just the same as if one was cureting the conjunctiva of the eye. When you are doing work in a tissue out of the range of vision, just say to yourself, What would I do if it were spread out before me? No one would think of scraping out the mucosa of the uterus if he could see what he was doing. Just because he cannot see it he scrapes away at it regardless of the mischief he is doing. It is just the same in speaking of pelvic infections. If we could open the pelvis and spread its viscera and peritoneum out on a flat surface, we could see what changes take place and what organs are involved in the infection. But, as we cannot see them, we should have a mental picture of the pelvis, its organs, the foci of infection, and the lines of transmission from the primary infection in the various pathologic processes. The lesions are similar and just as clean cut and consigned to definite tissues as are infections of the structures of the extremities. One is a cellulitis; the other is an ulceration or an erysipelas, etc. Take, for instance, a streptococcic infection that follows parturition. That is a subperitoneal erysipelas in the pelvic cellular tissue. What have we been doing in those cases? We have been taking out the uterus, the ovaries, scraping and cureting the uterus for these infections. We would not think of cureting or amputating the fingers for an erysipelas of the arm. If you curet, you will spread the sepsis. Just follow an infection in the subperitoneum. The infection has extended into the cellular tissue and the lymphatics under the peritoneum from the uterus. The lymphatics carry the streptococci along behind the peritoneum and in front of the vessels and nerves in the pelvis, never in the peritoneum; therefore, the infective material is beneath the peritoneum. It is a subperitoneal erysipelas, and no amount of drainage of the peritoneal cavity will cure the infection, because the infection is behind in the cellular tissue. You must open into the infection and drain.

We will open this abdomen along the line of the old scar, which we will later excise before we make the final closure. I am endeavoring to get into the free peritoneum first above the line of adhesions, and then attack the adhesions, so I can get some idea of where I am. I take it, from the way this peritoneum comes up, that it is not adherent to the bowel at this point. I was right. One should have a definite plan of procedure in all intestinal fistulas, namely, you must free the intestine from its attachment to the parietal wall. That is one of the important things. On inspection of the pelvis there is the ovary, I take it, right there. It feels like it. There is the tube; the fimbriated end is free, notwithstanding all that inflammatory condition in the pelvis. That is a great relief to find these, because it helps me to get some light through here and obtain anatomic landmarks.

There is a mass here behind the uterus that feels firm and hard. It is composed of intestine. How many coils of intestine are in it? I do not know, but we must free them. I am going down around the site of the abscess that was over on the left side and behind the uterus. You can see how these intestines lined up into one point of fixation. There is an abscess down below—one of many, I presume. Now I will examine and free one coil of intestine after another. That must be done very carefully. One coil goes down to the central pocket of the suppuration. There is an opening in the intestine. There is a second coil. There is also an opening in it with organic adhesions where these two coils came together. I am endeavoring to get them free. I will see whether there is any leakage elsewhere. No, there is not. That coil is out of the field. Here is another adhesion. There is another coil out in the field. Now we come to two coils There is one. There is a dilatation of the bowel here, meaning that there is an obstruction below. Here is the other end that is small and contracted—below the point of cicatricial obstruction. The obstruction is caused by a band of adhesions which we must free. There is the mesentery. What one endeavors to keep away from in this work is mesenteric vessels. When you get into the mesentery, you get the bleeding and then you have real trouble.

Now we have freed this bowel down to one pivotal point. Here in the uterine fundus is evidently the place where the intestine was drawn into the uterus and where the uterovaginal fistula existed. I must endeavor to get that free. There was never any opening into the bladder.

Now we are down to the central point of fixation, and there is where the intestine is a part ot the cloaca in which the abscess is situated. There is an opening in the bowel you see. There is the intestine, there is the uterus. Now I have it free. Let me see whether it is one and the same coil. I will take it out into the field and see if it is. Yes; it is. You have got to get to the place where the mesentery is continuous. There that mesentery is continuous. That adhesion had to be split before I was sure that these were the two ends belonging to each other; they do belong to each other: this is the proximal and this is the distal end. There has been some operation done here. I do not know what it was, but this mesentery has been plicated. Here the mesentery was ligated. There is the mesenteric suture. We have to get these two ends of bowel united. We will unite them end to side. This is the proximal portion because it is the dilated portion, and that is the distal portion because it is the contracted portion. We will unite them end to side after resecting the area with the perforations.

I am preparing for the resection. The portion of bowel to be removed is drawn through the incision, and the peritoneal cavity is protected by gauze packing. The loop is emptied by stripping with the fingers, and clamps are placed on the bowel on each side of the proposed incision. The mesentery is then ligated a short distance from the bowel margin and divided.

Great care must be exercised not to cut off the blood-supply of the bowel which is to remain. We divide the bowel between the clamps with the actual cautery. We will close the end of the distal bowel. First we put in a catgut suture that goes through all the coats—a Lembert stitch. It just turns in that top, the same as you would invert an appendix. Then we put in a serosa suture. This does not go through all the coats of the bowel. It makes an inversion of that stump. Now I will come back and make a second inversion and get an overlapping of the peritoneum. That is the distal portion of the bowel. I know that is the distal portion because it is the contracted portion. The obstruction, you see, has dilated the proximal portion. That completely closes that loop of the bowel. Now we will unite the end of the proximal loop with the side of the distal loop. The anastomosis is made in the same manner as is a gastro-enterostomy. The serosa of the open end of the bowel is sutured to the serosa of the lateral wall of the distal loop, a sort of posterior row of sutures. Then the wall of the distal bowel is slit open with the scalpel; the edges of the proximal loop are inverted into the opening in the distal loop and the opening is closed all the way round by a complete Czerny-Lembert stitch, so as to prevent hemorrhage and leakage. The approximation is done very quickly. An outer row of serosa stitches completes the closure. I would have liked to have performed that operation with the button. It would have saved considerable time, but with all these adhesions here I feel there might be some question about its passage subsequently. Finish the operation by uniting the overlapping layers of mesentery with a few sutures to prevent any dangerous opening being left behind into which a coil of intestine may later be herniated.

One of the dangers of every suture operation on the bowel is the danger of hemorrhage into the lumen of the bowel. The Czerny stitch is supposed to act as a hemostatic stitch in the suture operation. We put in a row of stitches that was continuous for the entire circle and including all the coats. Then we made an inversion of that row of stitches. Remember that the weak point of every suture method of intestinal approximation is where the mesentery is attached. The long period of obstruction that had existed here has very much thickened the upper portion of this intestine, so that the wall is three or four times as thick as it should be.

Now that takes care of one section. We still have this lower condition to contend with. That has only one loop, and that is apparently the loop where the former resection was made; now that coil is free and out of consideration. Now we must commence to deal with this other coil. Let us hope that this is nothing more than an agglutination, because this is the sigmoid and there should be no leakage into that. From the story of the patient there should be no communication with the sigmoid, and we believe that is true because, when we made the methylene-blue enema test, none of the solution came through the fistula. Let us see if it is justified by the findings here. Here is the proximal limb of the sigmoid and there is the distal limb. The sigmoid is adherent to this mass. There was a sinus that opened down to the point of leakage. It does not appear to go through the intestine, but appears to be on the side of the intestine. If that is true, we are fortunate. You cannot distinguish the lumen of the intestine here because there is so much granulation tissue. It is just the granulation tissue of the sinus, I take it, and that there is no perforation of the bowel. I was right. That shows beautifully. See the aid we have had from the fact that the methylene-blue solution did not come through the fistula.

We will top-sew the weakest portion of this large bowel with chromicized catgut. We will simply turn in this sinus to strengthen the weakest portion of this large bowel. There was a cloaca or pocket here, so that if I put silk in this septic field it would form a sinus and I would have to take it out later. If I put in linen, I will have to take it out later. That is why I am putting in this chromicized gut, which will eventually be absorbed. That fistula is just a connective-tissue tract, and we do not have to take that out. That takes care of itself.

Let the record show that there were conditions here which showed that the small intestine had been divided at one place, and that there had been a plication of the mesentery and a fecal leakage into a cloaca or pus-pocket; that on the side of that was an adhesion of the large intestine; that there was a stenosis of the small intestine at this point with a dilatation of the coil above; that we freed the coil, resected a portion, and made an end-to-side union of the small intestine with suture; that then we freed the large intestine, which was firmly adherent.

That completes this portion of the operation and we will now close the abdomen. A drainage-tube will be placed down into the pelvis. We are not going to make an ectropion of the cut edges of the peritoneum because it has no value, where, you see, the adhesions existed from this point down. We are using chromicized gut to unite the muscle aponeurosis because there is a certain danger of infection occurring. We ordinarily use plain catgut for the aponeurosis. She lost but little blood, comparatively speaking; therefore we are not going to give her an intravenous saline infusion. She will be put in a sitting position, with the body almost upright, and we will not give her salines by rectum. We feel much hindered by that fact in this class of cases, because we would like to give her nutrient salines for the next five or six days. She will have just a little liquid by mouth, but given frequently. The skin wound is closed with horsehair, dusted with bismuth subiodid, and sealed with collodion gauze.

Let the record show that Sister reports the sponges counted and correct.

[The wound was dressed on the fourteenth day. There was a complete primary union down to the tube; no suppuration; no fecal discharge whatever since operation; tube removed. The subsequent course of the case was uneventful. The patient left the hospital at the end of four weeks, entirely well.—Ed.]

TUMOR OF AXILLA; FALSE ANEURYSM OF AXILLARY ARTERY, RESULT OF ULCERATIVE SYPHILITIC ENDARTERITIS, WHICH PERFORATED THE WALL OF THE ARTERY

HISTORY

Patient, male, aged thirty-five, enters hospital because of almost complete motor and sensory paralysis of right arm, hand, and fingers, and a tumor in the axilla.

Trouble began March 1, 1913, with a numbness in the thumb and index-finger of the right hand. Next morning the whole hand was very painful and numb and partially paralyzed. By the end of a week the numbness, pain, and partial paralysis extended to the elbow, and in four weeks the whole arm and part of the shoulder were very painful and almost completely paralyzed. About May 5, 1913, in the evening, the pain in the arm suddenly ceased and has not returned since. Six weeks after the trouble began the patient first noticed a tumor mass under the upper pectoral edge of the axilla, near the scapuloclavicular articulation. The tumor grew rapidly in size, and in four or five days reached its present size (size of a man's fist). He says it felt as though something "broke" there when he first noticed it. The tumor is soft to the touch, not painful, and not movable. The arm is swollen slightly and of a bluish, deep cyanotic color.

Patient had typhoid when a boy; gonorrhea three or four times when about twenty years old. Had a single sore on penis fourteen years ago. Doctor told him it was syphilis and cauterized it. Patient took "blood" medicine for two weeks. Says he never had a sore throat or rash, but occasionally had sores on tongue and mouth for ten years.

COMMENTS AND OPERATION

Dr. Murphy (Operation June 5, 1913): When did this paralysis first appear?

Intern: About April 6, 1913.

Dr. Murphy: You said the pain all ceased on May 5th. The arm was completely paralyzed when?

Intern: About five weeks after the trouble started.

Dr. Murphy: What relation did the pain in the arm bear to the paralysis? That is important. The pain first appeared about March 1st. The paralysis began about a week later. The pain ceased suddenly on May 5th. Did he have motion in his arm on May 4th? How long before May 4th was sensation absent in the arm?

What I want to find out is, is this a paralysis of motion and of sensation and did both occur at practically the same time? Did he have sensation to touch after that time, and if so, how far down? If it was a paralysis of sensation, where was the line of limitation of sensation? Is this a tumor in the brachial cords or plexus, or is it on the trunks back of the brachial plexus, or in the nerve-trunks below the brachial plexus? Or is it a tumor pressing on any or all of these structures? Is the musculocutaneous nerve involved?

Intern: Yes, sir.

Dr. Murphy: The musculocutaneous is involved—how do you know?

Intern: Because he has a complete paralysis.

Dr. Murphy: Of what?

Intern: From his elbow down.

Dr. Murphy: What muscles below the elbow does the musculocutaneous supply?

Intern: The flexors.

Dr. Murphy: No, it does not. What does it supply below the elbow? What does it supply above the elbow?

Intern: The musculocutaneous supplies the coracobrachialis.

Dr. Murphy: And what else? The greater part of the brachialis anticus and the biceps. Below the elbow it is merely a cutaneous nerve. What would that have to do with the motion below his elbow? Nothing. Can he swing his forearm up behind his back? No. That means that his long subscapular is out of commission. What about his internal cutaneous? There is a paralysis of sensation in the anterior internal and posterior internal. There is a paralysis of sensation and motion in all the nerves below the area supplied by the cervical plexus and by the external anterior and internal anterior thoracic nerves. There is only one position in which pressure could cause this type of paralysis and that is within the brachial plexus immediately at and extending upward from the formation of the median nerve, exactly where the second and third portions of the axillary artery are situated. This is an extraordinary case. I have never seen a case like this. Before he had the tumor, before he had anything of any importance in the way of swelling in the axilla, he had nerve symptoms in the hand and forearm, the numbness, which makes it look as though it is a lesion beginning in the nerve itself or close to the nerve, which compromised and compressed the nerve. That compression does not look right, because we have never seen as rapid a course as that from a compression *neoplasm* that did not involve the nerve-trunks. Now, then, if it were a tumor involving the nerve-trunks, how did it come to involve all of the large nerve-trunks simultaneously? It should not do that. If you will look at the chart (see Fig. 231), you can see at a glance the origin of these nerves. The median nerve supplies all the superficial muscles on the front of the forearm except the flexor carpi ulnaris. Its anterior interosseous supplies the deep group of muscles, except the internal part of the profound flexor. The median nerve is made up of branches from the outer and inner cords and lies on the anterior side of the third part of the axillary artery. The posterior cord is behind the second part of the axillary artery at this point. The inner cord is to the inner side and passes across its internal surface. The outer cord bears the same relative relation to its external surface. The brachial plexus is on the external posterior side of the first portion. If this man has an aneurysm of his axillary artery in its second portion, it could involve any or all of the nerve divisions that are paralyzed. We do not know when his radial pulsations ceased: they are absent now. He has no pulsation in the tumor and there is no bruit. An arterial aneurysm should pulsate. He has a venous stasis as well, which is the result of pressure. Is it, therefore, a lesion of the axillary artery or a lesion of the nerve- or cord-trunks? Finally, after the paralysis was complete, he commenced to develop a rapidly growing tumor in the axilla. He has an intense cyanosis of the arm, a suspension of pulsation, and the arm is completely paralyzed.

If it is a primary neoplasm in the nerve, that is, if it is a neurosarcoma, there is not a particle of use amputating that arm. Every one of those cases recurs, and every one terminates fatally, even when you have a small neurosarcoma in one of the nerves of the forearm. I remember reading, thirty years ago, the history of a man who was hooked by a cow. The pressure on the median nerve caused a sarcoma. The nerve was taken out, and finally the arm was completely amputated. That started me to think about it, and since that time all the cases which I have observed have gone to a fatal termination. Rapidly growing sarcomata or sarcomata of the nervous system that occur in that spot all recur. The clinical course of a neurosarcoma is that it is very slow of growth, but it is wickedly malignant.

Is this an aneurysm of the axillary artery, or is it an aneurysmal sarcoma? The aneurysmal sarcomata that occur in this position are very malignant. When incised, they bleed exactly like an arterial aneurysm and they terminate fatally very rapidly. As to aneurysm of the axillary artery, there is no bruit and no evidence of pulsation; this might be accounted for by coagulation having taken place within the sac. I cannot make the differential diagnosis now. I must expose this tumor to view. But what must I provide for first? I must provide for a place to retreat to when that blood shoots up over my head if this is an aneurysm. I must have a place above the clavicle where I can tie a ligature and stop the bleeding just as soon as I reach a place of danger. If I go in below and get on the axillary artery without having a retreat, a place of safety, the patient will probably die. If I first expose the subclavian above the clavicle, dividing the clavicle, if necessary, and put a provisional ligature on that vessel, I can safely open up all the axilla, and be ready to retreat to this place of safety when the hemorrhage begins.

Let me see if I can feel any pulsation in the axillary, brachial, or radial arteries. No; there is no pulsation to be felt anywhere. The axillary artery is compressed against the humerus. I think that is due to this mass in the axilla. The clavicle is crowded upward and forward. There is only one situation where the subclavian artery can be reached for digital compression—where it crosses the upper surface of the first rib. The third part of the artery is most favorable for ligation. Ordinarily, this can be done fairly easily, but in this case the clavicle is displaced upward, which naturally increases the difficulty of the operation. We draw the skin taut over the clavicle and make a transverse incision about an inch in length.

All these veins and arteries are dilated. Every little vessel bleeds, many of them severely, the result of increased collateral circulation. We ligate them all. The next step is to expose the clavicle and divide it. I have to take more than ordinary precaution in dividing this clavicle, because I do not know just how close to the clavicle this dilated blood-sac may be. I divide that clavicle with a Gigli saw, which is passed under it. I will make a double division, so as to remove about an inch of the clavicle, shelling out the bone from its periosteum. I now divide the subclavius muscle and expose the subclavian artery. I pass two silk ligatures around the subclavian artery, which is right in the field now, and secure the ends with hemostats, but I do not tie them. We are ready for an emergency now.

The next step is to attack the tumor. We make an incision along the anterior margin of the axilla, extending from an inch below the acromion outward and downward five inches. Retracting the skin-edges brings the pectoral muscles into view.

The next step is to take off the pectoralis major near its inner angle or costal attachment and turn it out of the axilla entirely. I make the incision here, because the nerve-supply to the pectoralis major all comes from the humeral side and not from the costal side. We will have a great deal of bleeding from the small vessels, and we will want a great many hemostats. I want to get away inside the axilla and go under the pectoralis minor muscle, which is now shining beautifully in this field. I am going to the inner side of the tumor, dissecting very carefully with my fingers. I believe this mass is a false aneurysm. It feels like soft rubber. We will open into it carefully. If it is a false aneurysm, it contains clotted blood. There is the blood coming out now—an old clot. Dr. Norman Bridge (visiting) will recall the day when Dr. Lee opened one of these tumors in the pharynx in his office and it was a dissecting aneurysm from an arterial leakage of the internal carotid. The man was dead in sixty seconds.

There is practically no bleeding from the mass. The bleeding is more from the capillary vessels that have endeavored to supply the arm on account of the occlusion of the main trunk of the artery. Now you will see the wisdom of the preparation above. We can stop this bleeding by compression. This is an aneurysm—a false aneurysm. There is a hole, $\frac{1}{3}$ inch in diameter, in the axillary artery, just below where the median nerve comes off. This is probably a case of syphilitic endarteritis. When the wall of the artery was ruptured, the blood was poured out into the tissues and formed this false aneurysm, which compressed the veins and the nerves, causing the cyanosis of the arm, and, by compressing the brachial plexus, it caused the numbness and the paralysis. I will have to ligate the subclavian. It is an ulcerative endarteritis. This is an unusual case.

I cannot make an arterial suture because of the condition of the wall of the artery. Therefore we will ligate the axillary artery above and below the perforation, and also tie the two ligatures on the subclavian. We will tie all these vessels that bleed, on account of the rapid development of the collateral circulation. I have not tied the subclavian yet. This man will have a fairly good chance to save his arm. Why? Because he has already had an opportunity for the development of a collateral circulation.

That is a very happy outcome, because I thought it might possibly be a sarcoma. If he gets a gangrene of his arm, which is possible, we will be able to amputate it and save his life. However, I think a sufficient collateral circulation will be developed to save the arm.

Let the record show that there was a circular perforation of the artery, about ⅓ inch in diameter, just above the position where the median nerve is formed; that this perforation produced a dissecting aneurysm, which caused a compression of the brachial plexus and the sudden paralysis. It was not a true dissecting aneurysm—that means, within the coat of the vessels. This was without the wall of the vessel. It perforated the vessel. It is probably of spirochetic origin, because he has had syphilis, which must be taken into reckoning all the time.

In the history it is noted the patient said that "it felt as though something broke." I think that is a very good description. That artery perforated as the result of an ulcerative endarteritis of spirochetic origin. That clears up the history of this case and also the secondary manifestations.

Now we are going to ligate the subclavian artery. The two ligatures are about ⅓ inch apart. I do not think these muscles will bleed any more with the subclavian tied. A gradual suspension of the circulation, as in this case, is what saves the limb from gangrene. Some years ago I wrote an article on blood-vessel surgery showing how practical arterial suture is in cases in which it is indicated. In the year 1896 I did the first successful end-to-end suture of an artery that was ever done. Arterial suture is just coming into vogue now. At that time we did three vessel sutures in succession, and all the patients lived, and all the extremities were saved; no gangrene. One was a case of end-to-end suture of the third portion of the subclavian. I first turned the muscles up and took the clavicle out, just as I did this morning, and then made the end-to-end suture. The other two were for perforations of the first portion of the femoral artery above the profundus. Dr. Neff looked these cases up about four years after operation and they were in perfect condition. All were for perforating bullet wounds with acute aneurysmal formation. Some of the men who are writing now have overlooked these cases. I read the paper at the Moscow meeting of the International Medical Congress in 1897.

Now, you will see, I have left a blood-supply of this axilla which should be ample. We will suture together the cut ends of the pectoralis major muscle. That muscle will be just as useful and as strong as before. I had a beautiful illustration of the division of the biceps, right through the middle, in a man who made his living by athletics. He had the largest biceps I ever saw. It was divided subcutaneously down to the bone. It happened in a railway accident. When I saw him six weeks after the accident I could put my fist in the gap in the biceps. He made an agreement with the railway company that if his biceps was restored to the strength it had before the accident he would not ask for damages, merely remuneration for his time. The cut ends of the biceps were not sutured. It healed well. At the end of a year he could lift many pounds more with that arm than he ever had been able to do before the accident, notwithstanding the fact that there was nothing but a mass of fibrous tissue connecting the two ends of muscle and no operation had been performed.

We will close both these incisions very carefully with horsehair and put a large dressing in the axilla to take care of possible oozing. The arm will be loosely bandaged to the side of the body in flexion, the hand resting on the chest.

[The patient made a splendid recovery. Primary wound healing; stitches removed on twelfth day. The arm had a natural color, showing that the collateral circulation was well established. There was no pain or numbness in it. Sensation was present, but slightly dulled; motion was returning very slowly. Pronation was restored on the fifth day. He left the hospital in about four weeks.—ED.]

TALK ON CANCER BY DR. W. L. RODMAN OF PHILADELPHIA

At the Clinic, on Thursday, June 5, 1913

Dr. Murphy: We are blessed today in having with us one of the master surgeons, one of the most accomplished teachers of surgery in the world, one of the men who is having extensive experience, particularly in relation to carcinoma of the breast. He is just the opposite to us—he is optimistic concerning the final results in carcinoma.

I have the pleasure of presenting to you Dr. William L. Rodman, of Philadelphia.

Dr. Rodman: It is exceedingly embarrassing for me to speak after so very flattering and undeserved an introduction by my distinguished friend, Dr. Murphy. He has said that he is a pessimist on the cancer question and that I am an optimist. I am. If cancer is a local disease, which assuredly it is, and was demonstrated as such in 1867 by Moore, of London, then why is it not curable? As long as cancer was thought to be but the local manifestation of a general or blood dyscrasia, then, of course, operative results were necessarily far from reassuring,—indeed, disheartening,—and were performed more with a view to relieving pain and prolonging life than with expectation of radical cure. Such procedures were incomplete, faint hearted, and performed upon advanced cases. Hence they were almost uniformly unsuccessful. A cure was unexpected, fortuitous, and accidental. I know of no pathologist who maintains that cancer is a blood disease. If primarily local, then why is it not curable if one only operates before metastases have taken place? The whole question then is to operate before cells have been loosened and swept from their primary focus along lymphatic vessels and to the neighboring lymphatic glands. If you could do that, then you will cure not only a majority of the cases of cancer, but practically all.

Is it not irrational to believe and teach that a purely local affection is, from its inception, incurable? What are the evidences pointing to the local origin of carcinoma? They are clinical, microscopic, experimental, and operative.

In the early stages of carcinoma, no matter where situated, there is no pain, loss of weight or appetite, sleeplessness—indeed, anything to indicate a constitutional affection. But the tumor grows, perhaps luxuriantly, and it is this, and this only, that causes the patient anxiety. The suspicions of many such patients will be allayed because they feel so well and are even gaining in weight. Could this be so if the disease were a constitutional one? So much for the clinical picture.

As suggestive as the clinical evidence is, the microscopic is well-nigh, indeed, positively, conclusive. If the growth is cut out, and with it the tissues adjacent thereto, what do we find? That the farther we get from the center of the growth, the fewer are the characteristic cancer cells, until presently none at all can be found, and the surrounding tissue is normal. Can this indicate other than a strictly local process? The experimental evidence is confirmatory. If mice are inoculated with carcinoma successfully, a small tumor forms at the point of inoculation—not many tumors, mark you, but a single one, and that at the exact spot of the injection. I never saw cancer in the human subject begin otherwise. It is always solitary, never multiple. Follow the mouse further. It continues in good health until metastases occur and then sickens and dies. In other words, it suffers primarily, as the human being, from an apparently local affection; later on, or secondarily, from general or toxic symptoms.

We have said that the clinical evidence is suggestive, the microscopic well-nigh conclusive, and the experimental, confirmatory. Discard them all, and no fair-minded jury will fail to find a verdict upon the surgical evidence, alone and unsupported. What is it? That as long as operations were incomplete and on advanced cases, radical cures did not occur. Secondly, when more extensive, we may say fairly radical, measures were practised, but still upon advanced cases, cures were exceptional and rarely encountered. But today, with even more radical operative measures upon cases as they present—some early, that is, with a local tumor, but no secondary ones or metastases; others fairly so, or presenting a moderate infection of the nearest lymph-nodes; others still very advanced, with extensive metastases to lymph-glands, mayhap elsewhere—more than half of such patients will be permanently cured. If the first group be considered alone, at least 80 per cent. will be cured; if the second, only 25 per cent.; if the third, *none*. If a single cancer cell is left behind, recurrence is inevitable. But some of you will say that my conclusions are general, and apply to no special group of carcinomata. Let us apply them. Take, for instance, the lip, where such lesions should be treated as radically as the one Dr. Murphy is, at this time, I doubt not, bringing to a successful issue. That is to say, the original focus, the lymphatic and submaxillary glands on both sides, with their intervening lymph-vessels and surrounding tissues, should be removed en bloc. There must be no possibility of leaving behind carcinomatous cells unless emboli have been carried to the bones or internal organs, in which event they will be overlooked and recurrence certainly occur.

At the Tübingen Clinic (1885–98) there were 66 per cent. of permanent cures. But the latest of these operations were done fifteen years ago—before dissections en bloc and removal of glands first *before* the primary focus were practised. It is not generally followed, though it should be, even now. It is much better to remove infected glands first, then the intervening lymph-vessels, and finally the original focus, in this way avoiding expressing cancer cells by manipulation or compression by either hands or instruments. Cancer of the lip should be diagnosticated and treated earlier, perhaps, than carcinoma when situated elsewhere, as it is superficial, seen every day, and there is no possible excuse for overlooking it. It begins as a crack, ulcer, wart, or leukoplakia, and very generally in males who smoke and are over forty, indeed, likely to be past fifty, years of age. If operated before metastases to the submaxillary or other glands have occurred, practically all such cases should be permanently cured. Rarely, if ever, have I witnessed recurrence in any patient who presented himself for operation before there was contamination of the neighboring glands. Per contra, advanced cases, especially when the bone is affected, mostly always are fatal. I have seen no permanent cure follow excision of the jaw.

Dr. Murphy, in his introduction, referred to cancer of the breast. What are the results here? They have improved decad by decad since Moore's teaching, in 1867, inasmuch as the Danish, German, and Austrian surgeons, who were the first to accept Moore's teaching, secured 9.05 per cent. of three-year cures during the decad from 1870 to 1880, while S. W. Gross and Sir Mitchell Banks reported 21.5 and 21 per cent. respectively in cases operated between 1880 and 1890. W. T. Bull, of New York, in 1895 reported 26+ per cent. of cures, and about the same time Halsted, 41+. The gain of each operator was deserved and based upon a freer and wider removal of tissue. I have seen them all operate. It is now nearly twenty years since the epoch-making papers of Halsted and Meyer, advocating a larger wound and a complete axillary dissection, to facilitate which the pectoral muscles should be sacrificed. Others applying the principles so forcefully emphasized by them have secured, in smaller series, it is true, and these private patients, even better results, among the number Sir Watson Cheyne, Dennis, and myself. Private patients come earlier, are more intelligent, and submit to operation more readily, and are, therefore, more favorable subjects. Of 64 cases operated at the Johns Hopkins Hospital before axillary involvement occurred, 80 per cent. were cured; of those with axillary infection, 25 per cent. Let us consider the stomach, where 35 per cent. of all carcinomata originate (Virchow). Is the almost general pessimism out of and in the profession justified? Unhappy has been my own experience of cancer of the stomach, for nearly all such cases are practically moribund when I am first called in to see them. The last case sent to me died less than a week after I saw her, and could not stand even a palliative operation. I have done but a few partial gastrectomies for cancer and *none* of these was successful, though some made good operative recoveries. Usually the most to be considered has been a palliative gastro-enterostomy, and of these I have done a number, with gratifying results, inasmuch as pain and vomiting have been relieved and euthanasia assured.

In November, 1911, before the Congress of Surgeons at the meeting in Philadelphia, W. J. Mayo read a paper in which he stated that of 90 cases operated at his clinic, 36 per cent. were well three years, and 25 per cent. five years, after pylorectomies for cancer. This is most encouraging, and shows what can be done if either a clinical diagnosis of cancer be made fairly early or ulcers excised at the propitious time, or *when undergoing cancerization.* It is, indeed, with the knowledge we now possess, next to impossible to make an *early* diagnosis of carcinoma of the stomach. By early I mean applying the same standard that we have done with the lip and breast, viz., a single focus only, and no concomitant metastases to lymph-nodes, liver, pancreas, intestines, or other structures. Accepting, then, what has been proved abundantly, viz., that cancer of the stomach usually, perhaps always, develops upon the base of a chronic ulcer, and, furthermore, that when it can be diagnosticated clinically operative measures avail little, it is manifestly our duty to forestall it by operating in the *precancerous* stage. Not only is such a course warranted on account of potential cancer, but, as I endeavored to show in 1900, is actually the best treatment for ulcer itself, as by excision, and it only, can the dangers of hemorrhage, perforation, subphrenic abscess, hour-glass stomach, etc., be absolutely prevented. Gastro-enterostomy, valuable as it is in some instances, falls short of preventing with certainty any of the above complications. The lesion remains, and no one can foretell what will come of it.

Not only is carcinoma insidious in its onset when attacking the stomach, but everywhere. The only certain way to defeat it is to prevent it, coming, as it ofttimes does, with such silent tread that it is overlooked until fatal metastases have occurred. Surgery has accomplished much when we can promise 25 per cent. of cures in gastric cancer, 50 per cent. in those affecting the mammary gland, and upward of 75 per cent. of labial carcinomata. But our work as surgeons is not done: it has, indeed, just begun. True it is that operative measures have been carried as far as they well can be with safety. How, then, you may ask, can it be accomplished? First, by recognizing ourselves that there is nearly always a precancerous stage, if the danger-signal is only observed, and then pointing out to physicians that to delay in advising surgical interference at once is to assume an unwarranted responsibility. A wart, mole, or nevus on the skin which becomes active in patients after forty usually means cancer; hence have it out in time. A leukoplakia of lip, tongue, or mouth, particularly in those who smoke, sufficiently often—I will not say always—results in epithelioma as to justify immediate removal. Lacerated cervices should be repaired, for it has been clearly shown that nearly all cervical carcinomata follow lacerations, and that unmarried and unfruitful women, while as frequently suffering from corporeal cancer as multiparæ, are exempt from the cervical variety.

Abnormal involution in the breast is so frequently the forerunner of cancer as to demand, in advanced cases and in women who have passed the childbearing and reached the cancer age, ablation of the affected organ. Nothing short of removal is safe. And so we could go on, but I will not trespass upon your patience to do so, in order to show how cancer may occur in the tip of an appendix; around acquired or congenital diverticula in the large intestine; in the gall-bladder as the result of irritation and ulceration by gall-stones; in the kidneys as the result of calculi; in the urinary bladder following papillomata; in the prostate affected with hypertrophy and hyperplasia; and in the glans penis when there has been marked phimosis.

Before concluding my remarks I must answer the question of Dr. Bridge, who wishes to know my views as to the cause of cancer. Unfortunately, we cannot say what is the exciting cause of cancer, or whether it be extrinsic or intrinsic. It may be extrinsic or due to a parasite, but this remains to be shown. Though an agnostic, I am willing to be convinced and admit that there are many reasons for thinking that the disease may be of parasitic origin. But this much is certain, entirely beyond the realm of speculation, and therefore should be kept constantly before us, viz., that continued irritation resulting in chronic inflammation of an epithelial surface *practically always precedes cancer and is therefore the predisposing cause.* Such conditions, in elderly subjects particularly, are a perpetual menace to their hosts, and demand, as we have already said, surgical interference. It is better to prevent than cure any disease, and true as it is of all other affections, it is most true of cancer. Only a few days ago a gentleman from North Carolina was brought to me by his physician, Dr. McIver, on account of an ulcer on his lip which had lasted for a month and had failed to heal under treatment. We promptly excised it, believing it potentially malignant. The microscopist reported it as benign. I congratulate his physician upon his good judgment and prompt action. The patient is happy that he hadn't cancer, and I not unhappy that it was prevented. Instead of chagrin, when such reports are received from the laboratory, there is cause for exultation. The more of them the better.

I cannot sit down without expressing my obligations to our distinguished host for the unexpected privilege of speaking to you from this arena, which he has made famous the wide world over, drawing American and foreign surgeons to witness, as we have done today, his brilliant technic and achievements in the surgery of bones, joints, nerves—indeed, everything; and teachers to listen to his remarkable, forceful, and systematic instructions that they may themselves the better teach. I have learned this morning that he is soon to be honored with a Fellowship in the Royal College of England, and all American surgeons will agree with me that a more fitting honor could not be more worthily bestowed.

I thank you, gentlemen, for your attention.

Dr. Murphy: One would know from the doctor's closing remarks that he was bred and trained south of the Mason and Dixon line, where generosity in expression and courtesy may freely surpass the truth. His primary propositions on carcinoma are easily acceptable and accepted by me. What we have intended is that cases of carcinoma referred for operation and operated give us unsatisfactory results. Dr. Rodman states that if they were all operated early, they would all be cured, which we accept as probably true; but *what is early in cancer?* That is a question which I have asked in this clinic innumerable times and have defined as—early is the time in the development of cancer between the primary penetration of the basement membrane by the malignant cells and its first transmission into the neighboring lymph-channels or glands or less frequently its primary admission into the blood-stream. No one has determined, experimentally or otherwise, up to this date when that is. We presume, on splendid clinical evidences, that it varies very materially in different individuals and in different ages of the same individual, and also in different locations of the same individual, depending on the richness of its lymphatic supply and on its subjection to trauma or irritation—pressure, etc.

I concur most heartily in the sentiment of Dr. Rodman that the precancer stage is a still more desirable time for operation, a fact which I brought out in my paper before the Philadelphia Medical Society preceding the Congress of Physicians and Surgeons held in Washington that year. It was discussed by Sir Berkeley Moynihan and W. J. Mayo. The able talk of this morning by Dr. Rodman should stimulate us all to greater endeavor for an earlier and more radical procedure.

August 31, 1913: When the above clinic was given I was not cognizant of the splendid work in this line conducted in the Collis P. Huntington Hospital for Cancer Research, under the able guidance of Dr. E. E. Tyzzer and reported to me by his associate, Dr. Thomas Ordway. They demonstrated experimentally and for the first time a definite time for metastasis in the Japanese waltzing-mouse cancer. It was with great uniformity on the thirty-ninth day after inoculation. They demonstrated further that immunity to cancer developed on the cross-breeding of the Japanese waltzing-mouse and further that, while the Mendelian law was fulfilled in the gross appearance, anatomic and color-formation, in their following cross- and inbreeding the physiologic susceptibility to cancer inoculation was not restored in any of the progeny. These two facts, repeated from conversation and not from print, which may admit of some errors, are the two greatest steps in advance made in the cancer problem in the last half-century—indeed, since Virchow's cellular pathology was established. These observations open the gates to a vast new field of research and are a monumental dividend on the Collis P. Huntington Research Fund. These facts may hasten the time when we can form an estimate of the date of metastasis in carcinoma in man, and thus give us some idea of the time when a carcinoma ceases to be a purely local disease and therefore readily curable by extirpation.—J. B. M.]

SURGICAL CLINICS
OF
JOHN B. MURPHY
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TUBERCULOSIS OF THE LUNG; PRODUCTION OF ARTIFICIAL PNEUMOTHORAX BY INJECTION OF NITROGEN ACCORDING TO DR. MURPHY'S METHOD

HISTORY

PATIENT is a female, married, aged thirty-two years; enters hospital because of soreness in left side of chest, which she says is located a little to the left of the vertebral column, and about in line with the angle of the scapula; says there is no shortness of breath.

Past History.—First noticed the trouble in August, 1912. Caught cold; had no chill; does not know about fever, and was never sick in bed. Had a slight cough. This cold lasted one month. In March, 1913, she states that she again caught a severe cold and has not been free from it since. States that she has had this soreness since August, 1912, but it has not been painful to the touch nor has respiration increased it. Coughed some at that time. Cough disappeared during May, 1913. Returned two weeks ago, and has raised a yellowish sputum with occasionally red spots in it. Today, she states, there has been more of a red tinge to the sputum than usual; says that she has had no fever daily, but states that she has had occasional chills and then a fever followed. About five of these attacks since March, 1913. July 3d had a chill, some temperature, and this was followed by profuse sweats. Has lost eight pounds in two years. Treated for bronchial trouble for one and one-half years. March 23, 1913, she went to a sanatorium, and returned to Chicago July 5, 1913. Gained seven pounds while there and felt much better.

Family History.—Husband living and well. No history of tuberculosis or carcinoma.

Present Trouble.—This patient came for examination in July, 1913. After having returned from the sanatorium she began to lose weight and appetite; began to have a continuous dry, hacking cough, which in a few weeks became productive of a yellow, and occasionally a blood-streaked, sputum in which tubercle bacilli were found. The temperature ranged from 99° to 100° F. in the afternoon. Night-sweats were occasionally noted.

Physical Findings.—Examination showed a poorly nourished, anemic individual; the skin was dry and flabby. The chest was flat, with limited respiratory excursion of right upper portion. Palpation confirmed the limited excursion and slightly increased vocal fremitus over right apex. Most marked posteriorly. Upon percussion an area of dulness was found just above spine of right scapula and in second interspace. With the stethoscope rough expiratory sounds were heard and areas of bronchial breathing and many râles were auscultated. The *x*-ray showed areas of fibrosis and small areas of consolidation. On July 25th 118 cubic inches of nitrogen gas were injected into the right pleural cavity. For two days following this the cough and expectoration were markedly increased and the patient complained of occasional sharp pains in right apex and in right subscapular area. At the end of one week the patient's condition had improved markedly, the cough and expectoration were decreased, and the temperature in the afternoon did not exceed 99.5° F. On September 10th the patient was again examined, and it was found that the pneumothorax had for the most part subsided. The cough and expectoration were increasing slightly. On September 27th 140 cubic inches of gas were injected into the right pleura, giving the patient very little discomfort and no pain. The cough was slightly increased, but the expectoration decreased, and after this was not streaked with blood at any time. The patient's appetite improved, and the general condition was greatly improved. The afternoon temperature did not exceed 98.8° F.

[*Note.*—Upon examination on October 1st the bronchial breathing was not heard, and only a few fine râles. The patient had gained weight and the cough had almost entirely disappeared.—Ed.]

COMMENTS

Dr. Murphy (September 27th): Tubercle bacilli are present in the sputum, and, worse than that, it is a tuberculosis with a mixed infection—a tuberculosis in the left lung. She has had two nitrogen injections, one in July and a second ten days ago.

Many types of apparatus have been devised, both in this country and in Europe, since our original introduction of the method in 1898. The apparatus we use at the present time is the same as was devised by us and used by Lemke fifteen years ago. The more simple the appliance, the more practical it will prove. A properly constructed apparatus should provide very low pressure on the escaping gas and a means of measuring its volume. The appliance shown in the accompanying illustration consists of a tank (A) containing the compressed nitrogen, the gage at the top indicating the pressure of gas in the container. The rubber tubing leading from this tank connects with a receptacle (B) which contains sterile water upon which floats the cylinder (C), which is open at the bottom. The gas passing into this cylinder causes it to rise, and the indicator on the graduated upright (D) shows the number of cubic inches of gas contained in the receptacle. The two stop-cocks at the top control the intake and outlet of the gas from the cylinder, whence it passes into the tube which connects with the needle. The pressure under which the gas passes into the pleural cavity is that which comes from the weight of the cylinder (C) and is always uniform.

This is the original apparatus we had made for the nitrogen injections. You will remember that in 1895, 1896, and 1897 we did our first original surgical work on the lung, and in the oration on Surgery delivered before the American Medical Association at Denver in June, 1898, we read a paper on Pulmonary Surgery and Pneumothorax as a treatment for tuberculosis.* After I read that paper I found that Forlanini, of Italy, had temporarily used the nitrogen gas, although not in the same way, some ten or twelve years before, and published a paper on the subject in an Italian journal. We learned later, however, that the idea was given birth in 1821.

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* See page 946 for reprint of that portion of Dr. Murphy's address referring to Artificial Pneumothorax in Tuberculosis.

In October, 1821, Carson, of Liverpool, published a paper detailing experiments on animals in which he produced artificial pneumothorax. He made an incision between two ribs of a rabbit and allowed air to enter the pleural cavity. The animal lay for a few seconds as if stunned by a blow, then was all right again. Five days later a similar opening was made in the opposite pleura of the same rabbit. The breathing became short, rapid, and laborious, and the animal was restless and so weak that it could not stand on its legs. It looked as if the animal would die; however, in two hours it began to recover, and in four hours was apparently normal. After five days there was still some impediment in breathing. The animal was killed, and the autopsy showed the diaphragm with a concavity toward the abdomen. The external incision had healed.

Two other similar experiments gave the same result. Carson concluded that one lung might be reduced to a state of collapse with perfect impunity. There is no record stating that he did any such operation on the human. By analogy, however, he concluded that if the lungs could be kept quiet, lesions in them would heal, as well as those in other tissues, and that in an abscess of the lung "the sides of the abscess are prevented from falling into a salutary contact by the powerful retraction of the surrounding substances." Hence he suggested reducing one lung into a state of collapse to place the abscess in circumstances favorable for healing. He furthermore suggested to reduce the lung to a state of collapse by opening the chest-wall. To obviate danger of bleeding or compression of the fellow lung he would admit a small quantity of air into the cavity of the chest at one time, and allow an interval to exist between the successive admissions which may be necessary before the lungs shall be brought to a state of complete collapse.

Then he purposed that this treatment might be used for pulmonary tuberculosis, which, he says, can be cured only "by mechanical means, or, in other words, by surgical operation." In support of this he referred to several instances in history

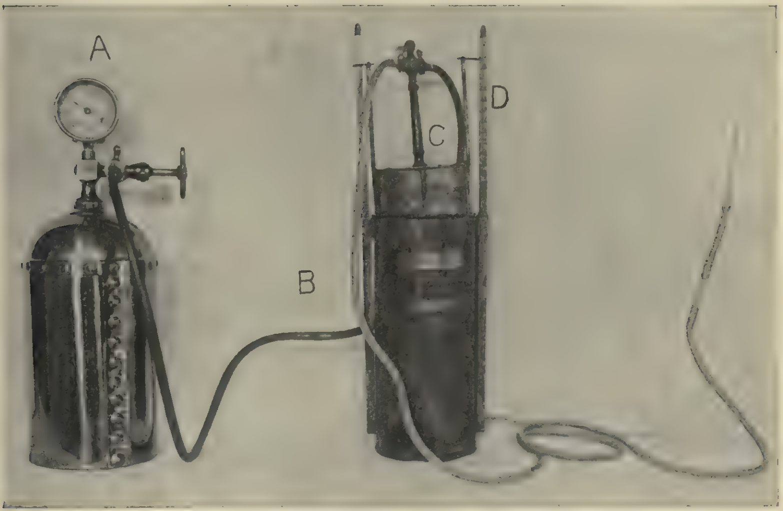

Fig. 232.—Apparatus designed by Dr. Murphy for nitrogen injection: A, The nitrogen tank with gage; B, tank containing sterile water; C, cylinder floating on water in B, and which contains nitrogen coming from A. This cylinder has no bottom; D, graduated upright.

where, in battle, soldiers received penetrating wounds of the chest-wall and were cured of tuberculosis.

Parolo (1849), Ramadge (1834), Canstatt (1843), Wunderlich (1856), and Echler (1867) all spoke of Carson's proposed treatment, founded on animal experiments, and conclusions based on clinical reports of the effect of accidental traumatic pneumothorax on tuberculosis, but there is no report to prove that any one of these men performed the operation on the human. Forlanini was the next to write on the subject of artificial pneumothorax, but never put the treatment into active use.

After delivering the address at the American Medical Association meeting, the lay press took it up and published this method of treatment of tuberculosis of the lung "as a cure." The result was that when I returned home, after a vacation in the mountains, I found my room packed three feet deep with mail from persons who asked about the treatment. The first time in my life that I was ever frightened was when I saw that mass of mail. We had given the treatment to patients before we read the paper. We demonstrated it at the Denver meeting of the American Medical Association, and had some splendid cases from the Chicago Tuberculosis Infirmary at Dunning, where we had been permitted to carry on our experiments. With the great demand from the public for treatment there was only one of two positions for me to take—either go into medicine and become an internist, or decline to give any treatments for tuberculosis of the lungs and remain in surgery. I chose the latter and personally never gave a nitrogen treatment for tuberculosis after that time. Dr. Lemke, who was a member of my staff at the time, took up the work and reported some 53 cases that he had treated. I see many of Lemke's patients now perfectly well—fifteen years after the treatment was instituted. In the period between the time of my original publication and his death he treated some 250 additional cases, making over 300 cases treated by him. Dr. James F. Neff, now of Spokane, and formerly with me, treated some 300 cases, and Drs. Golden, Hochrein and Kreuscher have treated up to date upward of 60, making a total of over 460 cases treated by those more or less closely under my observation.

It was the impetus given by my announcement in 1898, and not the paper of Forlanini, which influenced Brauer, professor of internal medicine in Vienna, to take up the work so actively, and he has continued this activity since that time. On returning from Europe recently I met Dr. S. J. Meltzer, of the Rockefeller Institute in New York. He reported to me that during his stay in Brauer's clinic Brauer told him that his own original paper was taken from mine. My paper was not the original paper. Carson's was the first, and Forlanini's the second, but neither man put the treatment into active use.

Since that time the German medical men and Continental Europeans have been using this method very extensively. How extensive this is may be judged from the fact that a short time ago I received a list of 384 published articles on pneumothorax as a treatment for tuberculosis of the lung. This list was collated from the literature, and shows how very wide-spread the use of the method is in Europe. A few clinicians in this country adopted the treatment for a time. Among these is Dr. Mary Lapham, of South Carolina. She took it up and kept it up, treating her patients on the same plan and with the same results that Dr. Lemke obtained.

The work has now become tremendously popular, and is used in most of the tuberculosis sanatoria of Europe. They are giving this method the precedence over others. It was one of the actively discussed propositions at the last International Medical Congress in London, and Professor Dock took the opportunity to say that it was introduced by me in 1898. Now, of course, that it has become popular in Europe, it will be accepted in this country, and we are sure that the treatment now will produce the results we claimed for it, and even better, as the public are becoming informed on the subject and the patients will apply for treatment earlier. Of course, Dr. Lemke's cases were nearly all late cases.

The time for giving the treatment is exactly the same as for treating surgical tuberculosis in any portion of the body, which means that it should be begun in the earliest stage of the disease, or, if possible, soon after mixed infection has occurred. When you have the patients come to you for treatment, often with an initial hemorrhage, often with the initial cough, what do you want to do? Treat it exactly as you would treat a case of tubercular hip disease, namely, *put the part at rest.* Pneumothorax is the simplest means of putting the part at rest, and is available and valuable in all the cases in which you do not have agglutinations of the pleura with the formation of organic adhesions. We believe the method of producing an artificial pneumothorax is most practicable in unilateral, apical, or monolobar tuberculosis in the early stages, as the pathologic conditions are such that the compression of the lung can be accomplished and adhesions are not likely to be found. It is not contraindicated where both lobes are involved, and it has been used successfully in many cases of this class.

Again, the earlier the treatment is begun, the better the prognosis. Every physician realizes that a disease in the apex of the lung will yield more readily to treatment than a lesion at the base, and that circumscribed foci are more readily affected favorably than scattered areas, even though the actual quantity of lung tissue involved in the latter does not exceed that in the former. Börninger, in 1912, said that artificial pneumothorax is indicated in all cases of phthisis with continuous fever and profuse expectoration if the patient's condition is not desperate.

Lemke reported good results in advanced cases. If both lungs are involved, the side most affected is injected first; the opposite lung is influenced by the injection through an improvement in the constitutional condition by the diminution of the absorption of toxins, lowering of fever, and cessation of sweats.

Börninger did not consider the procedure justified in the mild cases, which is exactly the opposite of what we originally taught and what we are now positively assured is the correct position to take. We do not consider that this method is indicated or practical in far-advanced cases of chronic tuberculosis where the fibrous tissue deposited in the lung will not permit of compression, nor where the pleuritic adhesions will not permit the gas injection. The dangers of injecting nitrogen into the pleural cavity theoretically were given as follows in my original paper: They may be—(*a*) A hemorrhage from wounding the intercostal vessels; (*b*) injecting gas into the intercostal vein, the possibility of which is questioned; (*c*) infection by the use of impure gas, infected trocar, or an improper antiseptic preparation of the skin; (*d*) rupture of infected foci into the pleural cavity through compression of the lung and separation of encapsulating pleuritic adhesions; (*e*) dyspnea from the use of too large quantities of gas; (*f*) puncture of the lung with the needle. This will not take place unless adhesions are present, and if the lung should be punctured, it is a matter of no consequence, as experiments and clinical experience show that the lung can suffer great traumatism without danger.

While these possibilities of danger are mentioned, no accident has ever occurred excepting in one case reported by Lemke (and he gave over 1500 injections), in which case he had an air embolism which caused a hemiplegia and the patient died from a combination of this and his tuberculosis a year or a year and one-half later.

The time for reinjection depends upon the gas used. From the experiments on dogs, we concluded that when air is injected into the pleural cavity, it is quite rapidly absorbed. This would tend to discourage the idea of attempting continued compression of the lung by means of intrapleural injections of air. Unverich demonstrated in 1896 by means of chemical analysis of the air injected into the pleural cavities of dogs that the different gases are absorbed with varying degrees of rapidity. Oxygen disappeared first, then carbon dioxid, and at last the nitrogen.

In connection with the first case injected with nitrogen that was reported in Denver in 1898, it was demonstrated, by repeated physical examinations and by roentgenography, that the lungs remained collapsed something over two months. In other cases reported at that time the absorption of the gas took place more rapidly, and it was Lemke's subsequent experience that in the majority of cases the absorption at the end of three and one-half to four weeks has progressed sufficiently to make the breath-sounds audible. From our more recent work we have concluded that the best results are obtained when the injections are repeated every two or three weeks, keeping the lung compressed during the process of cicatrization. What does the injection accomplish? *It puts the diseased part of the lung at rest.* It collapses abscess cavities, if such are present, forcing the contents of these cavities out through the bronchi. It gives the tuberculosis an opportunity to do what tuberculosis does every place where it repairs, that is, to form connective tissue, contract, and encapsulate.

Daus claimed that the compressed lung contained very little, if any, oxygen. He assumes that this may cause disintegration of bacilli into aërobic and anaërobic parts, and the sclerogenous toxins continue to be active, whereas the caseogenous toxins become inactive. Absolute experimental proof of this is lacking.

Bruns and Sauerbruch produced pulmonary fibrosis by ligating the pulmonary artery. The same procedure in the human caused a shriveling of the lung, supporting in a measure Stokes' claim that pneumothorax obliterates vessels, and hence causes atrophy and fibrosis. We believe, however, that the compressed lung, when relieved of pressure, expands and resumes its normal or nearly normal functions and size. Clinically, it has been noted that when two sides are injected separately in a bilateral case, the non-compressed lung functionates again. Daus contends, therefore, that it cannot be a question of fibrosis, although areas of fibrosis may exist, as shown by Forlanini. We believe that these fibrotic areas undoubtedly represent the former tuberculous lesions; therefore, the more advanced the disease, the more fibrosis, supporting our original contention for early treatment. We are pleased that Daus is following our plan of continuing the operation for months until the desired results are obtained, even for a period of months, if necessary. First cure the disease; then consider the reëstablishment of function in the diseased tissues, and in all the cases observed by us the lung expanded to fill the pleural cavity after the nitrogen injections were discontinued.

The method which we employ is the simplest one for injecting the nitrogen. It never causes a higher pressure than we should have. While other methods have been devised, we like this the best. If you employ the method requiring the use of water with the nitrogen, then you can get too high a pressure and you may do a lot of mischief. In our method the only weight is the weight of the cylinder. It never goes up and down, so that it is a uniform pressure all the time.

We always use the blunt needle with an opening on the side of the needle near the tip. This was my plan, and it was the plan used by Dr. Lemke. After the gas starts to flow you continue to let it flow until the patient complains of shortness of breath. With each inspiration the gas goes in and goes in until you get a compression of the lung on that particular side. Then you repeat this treatment every two or three weeks and keep the lung compressed during the process of cicatrization.

In following this plan Dr. Lemke found that, three or four days after the injections, the temperature went down, the purulent expectoration and cough diminished, the patient commenced to get an appetite, gained in weight, and improved generally.

Technic of the Injection.—The point of insertion of the needle varies somewhat in individual cases. If the apex of the lung be the seat of the lesion, the needle should be inserted in the fifth or sixth interspace, between the anterior and midaxillary line. The best point is the fifth interspace, at or near the anterior axillary line. If it be a middle or lower lobe tuberculosis, the injection should be made over the upper lobe, preferably in the fourth interspace, just outside the mammary line. Ethyl chlorid may be used for local anesthesia, but we have recently infiltrated the injection zone with novocain solution and have found it most satisfactory, for in this way the skin and subcutaneous tissues down to the pleura may be thoroughly anesthetized. A tenotome puncture should be made through the skin to permit of easy insertion of the needle. It is important, after the needle is inserted and before the tube is attached, to assure one's self that the point of the needle is in the pleural cavity, and this is done by instructing the patient to take a series of deep inspirations. If the needle is within the pleural cavity, a current of air may be heard to rush in. After several deep inspirations, if a little air has entered the pleural cavity, there will be current during both phases of respiration. Sometimes, even though the opening of the needle be within the pleural cavity, it is found that the current of gas meets with considerable resistance. This is due undoubtedly to the fact that the current of gas, unless much pressure be used, impinges upon such a small area of lung surface, and that it is not sufficient to overcome the cohesion that exists between the two layers of the pleura, which Lemke said had been estimated at 12½ mm. of mercury. If the needle has entered the lung, as it will when the two pleural surfaces are adherent, there is a slight current of air through the needle during the forced expiration and frequently a little frothy blood will appear.

The quantity of gas to be injected varies considerably, and should range from 50 to 200 cubic inches. The amount to be given in each case is best regulated by the symptoms of distress, dyspnea, and the displacement of the mediastinal contents and diaphragm. Börninger believes that gas may be injected until positive pressure is obtained in the pleural cavity. If the patient's discomfort is too great, the current of gas may be reversed until relief is obtained. The needle is then withdrawn, the wound sealed with collodion, and a moderately firm compress is placed over the puncture. The compress is to prevent the escape of gas from the pleura into the subcutaneous cellular tissue.

Börninger suggests that the subcutaneous emphysema might extend to the anterior mediastinum and heart. He thinks this may account for some deaths. We believe that he is in error. We have noted no such manifestations and have had no such deaths in any of our cases.

At the first injection we use a moderately sized, blunt-pointed needle which has a lateral opening near the tip. For the subsequent injections a smaller needle may be used, provided this puncture is near the site of the original injection.

In our original article we reported seven cases of pulmonary tuberculosis treated successfully by this procedure. Lemke, in 1900, reported 53 cases treated by himself, and an additional 12 cases treated by Thomas, of Spokane, Wash. Subsequently Lemke reported an additional 40 cases with gratifying results.

This makes a total of 114 cases treated prior to 1901. Each one of these cases received from 1 to 5 injections, the minimum amount being 50, and the maximum 170 cubic inches of nitrogen gas. In this connection let me say a few words about the immediate management of pulmonary hemorrhage. One can, by careful examination, determine the site from which it is taking place and produce an immediate compression of that lung by means of artificial pneumothorax, produced in the manner we have employed. It is exceedingly simple. Take an ordinary hypodermic needle, rub the sharp point dull on a brick, cover the butt end of the needle with cotton, which will serve as a filter of the air that is to enter, then insert the needle into the pleura at the point of election for the production of the pneumothorax. The skin should have been prepared previously by painting it with iodin and puncturing it with a tenotome, as is our custom. The idea now is to let the air enter the pleural cavity through the needle, the cotton filtering it as it enters, thus producing a pneumothorax. The finger placed over the butt end of the needle serves as a valve. As the patient inspires the finger is lifted off the needle to allow the air to enter, and on expiration the opening is closed with the finger. In that manner you can pump the pleural cavity full of air or to any desired degree of compression. If the patient becomes too cyanotic, or if the breathing is embarrassed, lift the finger from the needle and allow a little air to escape. The procedure is now reversed. Close the end of the needle with the finger on inspiration, and remove the finger on expiration, so that the air will be pumped out instead of in. It is a very simple but an exceptionally valuable procedure for the treatment of pulmonary hemorrhage. The instruments necessary are always at hand. The method can be instituted quickly, so that there will be no time lost. All you need is a blunt needle, a little cotton, and at the most some ice to freeze the skin, but even this is unnecessary, because when speed of operation is necessary, local anesthesia need not be resorted to in any of these cases.

I have already referred to Dr. Lemke's case of air embolism followed by hemiplegia from which the patient died. Whether it was an air embolism passing through the hole made in the vein I do not know, but I had the same thing happen in a case I aspirated for the determination of pus in the lung. I put in the hypodermic exploring needle to see if there was pus in the lung. The patient coughed following the withdrawal of the needle and suddenly had a convulsion. He got a hemiplegia, and died about forty-eight hours afterward with a high temperature—106° to 107° F., showing that septic material had been admitted from the pus cavity into the vein and carried to the brain. When I punctured the abscess this material went from the abscess into the veins and produced a hemiplegia. That is possibly what happened in Dr. Lemke's case. That does not matter. We charge it to an accident. That was the only accident that occurred, showing what a comparatively safe method it is.

It is evident, from the trend of pneumothorax treatment at the present time, that we were about fifteen years ahead of the times in advocating it in 1898.

It seems to me now that in tuberculin we have an additional aid, that is, when it is properly administered. I do not think there is any agent that is so erroneously administered as is tuberculin. There is no average dose, no regular dose, of tuberculin. Each patient who is treated with tuberculin has to be treated as an individual equation. What does tuberculin do? It increases the opsonic index. Can you do that without tuberculin? Yes, and decidedly so. Most beautiful is the work done by Patterson, at the Frimley division of the Brompton Hospital for Tuberculosis, in this line. When we made a tour of England three years ago the most striking results we saw were those secured by Patterson in the management of tuberculosis. He does not use tuberculin. He just uses graduated exercise and rest, and he uses it as no other man in the world uses it, and uses it to get results. First, he knows why he does it; second, he knows when he is giving a sufficient dose of exercise to get a proper opsonic reaction. He brought out chart after chart, a hundred or more, showing the patients who had come down from London with a temperature of 101° to 105° F. They were put to bed; in three, four, and five days their temperature was down to 99° to

99.2° F. What did he do to lower that temperature? *Just what we do not do.* There is no disease that is commonly so outrageously and badly treated as is tuberculosis, and is likely to continue so until the laity become actively interested, and the laity will shortly know more about its management than we do. We permit a tuberculosis patient with a temperature of 103° F. to walk around. The profession had the walking fad here for a time, and we even had patients walking barefooted in wet grass and climbing mountains before breakfast. What does this exercise do? It produces too much absorption of the autotuberculin, gives too high a reaction, and lessens the opsonic index.

How does Patterson deal with this problem? He puts that patient to bed; puts a nurse in charge of him; does not let him get up to eat; does not let him up to go to the toilet; does not let him read or have visitors; gives him an absolute rest, mentally and physically, *and his temperature is down to normal in five or six days* unless he has a pus retention cavity in his lung. Then he will elevate him in bed enough to feed him while he sits up. Then he will elevate him enough to let his feet hang over the side of the bed. Then he will let him sit up in a chair. The moment the vesperal temperature exceeds 99.5° to 99.6° F., which is the correct opsonic index temperature, the same as with tuberculin, the patient is put back to bed again. He gradually increases that exercise, getting the afternoon temperature along about 99.5°, 99.6°, 99.7° F., until we saw women out in the woods with great big hatchets grubbing out oak roots, *cured.* That has resulted from system, from dealing with them not on the Wright plan of producing an opsonic index, but on the rest and exercise plan of getting tuberculin from themselves.

It was not uncommon when we were first giving the nitrogen gas injection treatment, to have the patients come to the office, lie down on the table, receive the injection, and go home. I think it ought to be given with care in the patient's home or in the hospital, because then you can watch these patients until you are certain that all is well.

If you will look over my original article of fifteen years ago you will find some beautiful skiagrams, just as good as they are made today, showing the collapsed lung after the injection. If you give the injections often, you can use ordinary air. Nitrogen is used not because it has medicinal properties, but because it is absorbed more slowly than air. Ordinary air contains 20 per cent. of oxygen, which means that it is absorbed much more rapidly.

Since our original paper in 1898 a number of operative procedures for the establishment of collapse of the thoracic wall or forced compression of the lung have been resorted to, with varying success. If the patient's resistance is lowered by any operative intervention in the presence of an active tuberculosis, the disease is given an opportunity to advance rapidly. Thoracoplasty and other operations of this type as a means of treatment for pulmonary tuberculosis must ultimately disappear, although a few brilliant successes have been reported by Friedrich and others.

The additional treatment of all these cases consists of the most favorable hygienic surrounding and of physical and mental rest, as much as it is possible to obtain. The diet is wholesome and nutritious. Tuberculin injections are given once each week. We always use Lucius and Brüning's preparation of Koch's old tuberculin. We make four solutions or dilutions as follows:

Take 1 c.c. of the pure tuberculin, and dilute it with 9 c.c. of ½ of 1 per cent. carbolic acid solution. This makes Dilution No. 1, and is in strength 1 : 10.

Take 1 c.c. of Dilution No. 1, and add 9 c.c. of ½ to 1 per cent. solution of carbolic acid. This makes Dilution No. 2.

To 1 c.c. of Dilution No. 2 add 9 c.c. of ½ of 1 per cent. solution of carbolic acid. This makes Dilution No. 3.

Take 1 c.c. of Dilution No. 3 and add 9 c.c. of ½ of 1 per cent. solution of carbolic acid. This makes Dilution No. 4.

We use a Luer glass syringe. We do not keep Dilution No. 1 over two months. Dilution No. 2 may be kept thirty days; Dilution No. 3 two weeks, and Dilution No. 4 for a week. They must be kept in a cool place. At the expiration of the time stated the respective solutions are renewed.

We keep the solution in glass-stoppered bottles. Everything is sterilized before the solutions are made.

For the purpose of making the solution we would suggest obtaining the following:

1. A Luer glass syringe with needles.

2. 1 c.c. pure tuberculin, Lucius and Brüning's preparation of Koch's old tuberculin.

3. A 10 c.c. graduate.

The injection should be given in the afternoon, preferably at 4 o'clock. The temperature should be taken every two hours during the daytime for the succeeding twenty-eight hours. The best opsonic index is obtained when the reaction on the afternoon following the injection is 99.6° F. The temperature reaction is the guide to the size of the dose, which must be adapted to the individual case. If the reaction exceeds 99.6° F., the dose must be reduced 1 minim—or more—the next week. If the reaction is less than or just 99.6° F., the dose may be increased 1 minim the next week.

Ordinarily in pulmonary tuberculosis begin with 5 minims of Dilution No. 3 as a therapeutic dose, and increase or decrease the weekly dose 1 minim according to the reaction as stated above. When the weekly dose has reached 20 minims of Dilution No. 3, begin with 3 minims of Dilution No. 2, increasing the dose 1 minim until a 20-minim dose is reached. Then begin with a 3-minim dose of Dilution No. 1 and increase the weekly dose 1 minim until a 20-minim dose is reached. By this time the patient should be cured and immune. Please understand that this is a general outline, and must be adapted to the individual case in such a way that the reaction does not exceed 99.6° F. the afternoon following the injection.

The following case of Dr. Kreuscher's is another illustration of the type of case in which the nitrogen injection is indicated:

Patient, female, aged thirty-six, came to the office in July, 1903, on account of a persistent cough and some expectoration; tongue coated gray, slight afternoon temperature elevation, ranging from 99° to 100° F.; the sputum was examined and showed no tubercle bacilli. She was well during the following eight years. Patient returned to the office February 26, 1913, on account of a slight cold and a cough which had persisted for several months.

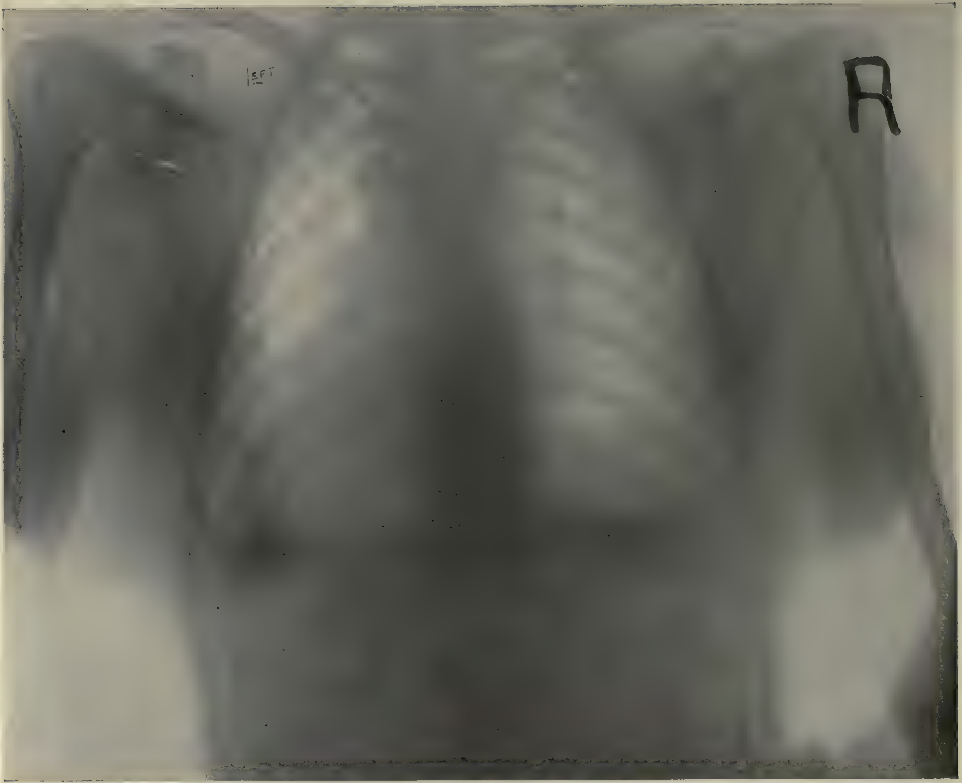

Fig. 233.—Pneumothorax. Tuberculosis of upper portion of left lung before injection of nitrogen gas. Diagnosis confirmed by tuberculin test, physical examination, and sputum examination.

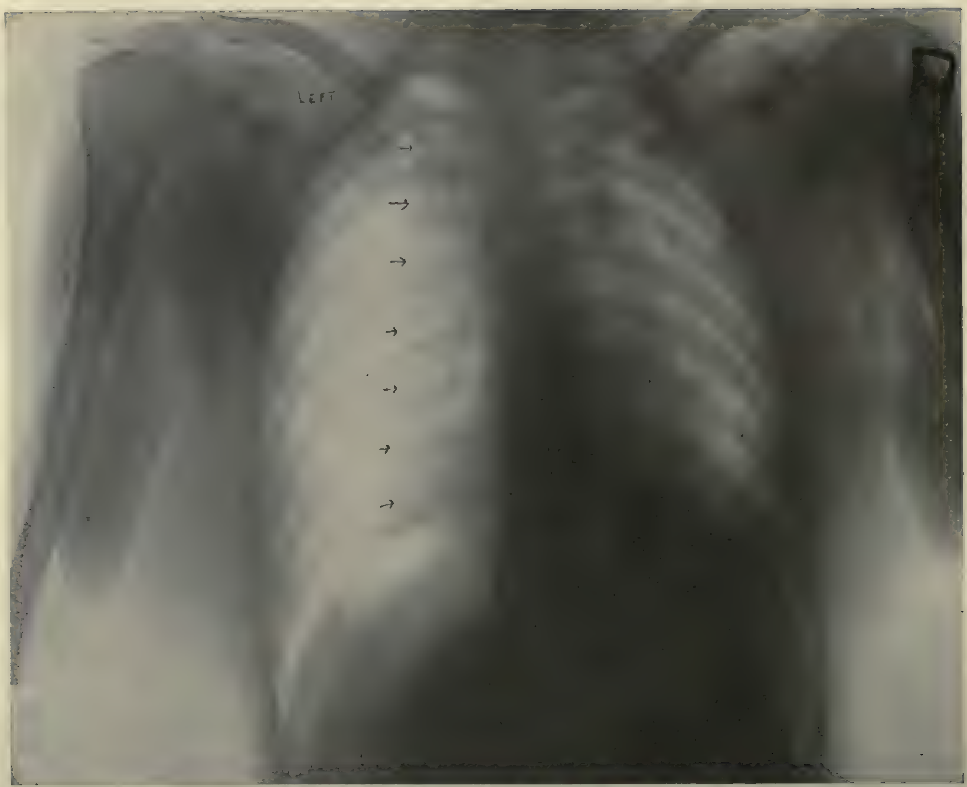

Fig. 234.—Pneumothorax. Compression of lung by injection of 110 cubic inches of nitrogen gas. Lung margin indicated by arrows.

Present Trouble.—After having seen Dr. Murphy in 1903, the patient was very much improved for a number of years. She had no cough and only an occasional slight cold. Two years ago, after patient had been working hard, she had a nervous breakdown and was always tired and worn out. A doctor gave her medicine and advised rest, and the patient improved for a time. However, in October, 1912, she developed a cough which has persisted to the present time. All this time there has been considerable expectoration, consisting of a yellow material, and on several occasions there was a bloody expectoration; the cough was most pronounced in the morning, and during the sudden temperature changes in the day-time. Patient says she has lost no weight, has no night-sweats, but her appetite has been poor for the last three months, and she feels tired and worn out all the time. Patient complains of "gas in the stomach" after eating, occasionally with distress in the epigastrium. She does not vomit, but in the morning, after coughing, often feels nauseated. Past history is negative. Father died of tuberculosis at thirty-one years of age.

Examination.—Patient is well nourished, has slightly hectic cheeks, but shows no sign of suffering or emaciation. An examination of the chest shows forced râles in the left apex. A tuberculin test dose of five drops of dilution No. 2 was given, with a temperature reaction of 102° F. twenty-four hours later. Patient was then put on the tuberculin treatment and received injections once each week for four months. Cod-liver oil emulsion with strychnin was given three times a day after meals in large doses. Patient received her last tuberculin injection July 13th, and then was sent up into Wisconsin for a vacation. She returned October 16th, and in spite of forced feeding and rest had lost three pounds. The cough still persisted. She feels tired the greater part of the time, and feels that she has a temperature in the afternoon. The temperature ranges from 99° to 100° F. each afternoon. The sputum is often slightly streaked with blood and contains tubercle bacilli. She was sent to the hospital, and on October 19th 110 cubic inches of nitrogen gas were injected into the left pleural cavity. (See Figs. 233 and 234.)

Dr. Ethan Allen Gray, Medical Superintendent of the Chicago French Air Hospital, has kindly sent me the record of one of his cases of pulmonary tuberculosis, in which he produced an artificial pneumothorax by means of nitrogen gas injected according to our method.

The patient, a female, was admitted to the Fresh Air Hospital June 17, 1913.

Weight on admission, 115½ lbs. Now, 135.
Temperature on admission, 96.8° to 100°. Now, 98° to 98.4° F.
Pulse on admission, 90 to 100. Now, 78 to 82.
Sputum on admission, 20 to 25 c.c. Now, 0 c.c to 10 c.c.
June 23, 600 c.c. N insufflated.
June 26, 600 " " "
July 2, 800 " " "
July 14, 775 " " "
August 2, 600 " " "
August 25, 500 " " "

In a letter to me, Dr. Gray says: "I am glad that you feel that you can make use of this, and hope to see a great impetus given to the pneumothorax therapy in the near future."

Abstract from the Oration on Surgery Delivered by Dr. John B. Murphy, at the Meeting of the American Medical Association, at Denver, in June, 1898

TUBERCULOSIS

Tuberculosis of the lung has been considered a medical and not a surgical disease. In a few instances the surgeon has had the courage to open and drain tubercular cavities in the lung; in a still smaller number he has excised a portion of the lung for the removal of a circumscribed tubercular nodule. Wm. Koch suggested the injection of disinfectants and chemical irritants into the diseased portion of the lung, but they have been used to a very limited degree. With these few exceptions, the disease has been left entirely to the care of the physician. We ask ourselves, what has been achieved in tuberculosis? With the exception of a thorough knowledge of its etiology and pathology, comparatively nothing. We have volumes of literature on the subject, describing its various phenomena, course, and terminations. From a sanitary standpoint, since Villemin in 1865 showed its infectious character and Koch in 1882 discovered the particular bacillus, we are possibly lessening the number of infected; from a therapeutic standpoint we had hoped, and still hope, that tuberculosis may be cured, alleviated, or curtailed in its destructive effect by products derived from the tubercle bacillus under certain conditions. The results barely justify a continuation of the hope. Can surgery contribute anything toward the repair of tubercular lesions of the lung? It has accomplished much in the treatment of tubercular lesions of the articular and osseous systems; its results in the treatment of tuberculosis of the peritoneum have been little less than brilliant. The ideal treatment of tuberculosis is eradication—extirpation of the tubercular focus. In only a few instances has the surgeon been able to attack it so as to obtain this ideal result, as in cutaneous, synovial and osteotuberculosis. His best achievements have been obtained in cases where he merely placed the tissues under favorable conditions for repair by nature, as draining of the peritoneal cavity, with or without extirpation of the original focus of infection. This necessarily demands the consideration of the reparative power of individual tissues when attacked by tuberculosis. Some tissues resist completely its invasion, as the fibrous tissue of tendons and fascia; others, when attacked, never automatically repair, as tubercular ulcers of the intestine. In still others, as the peritoneum, the reparative power or resistance offered to the advancement of the disease is great. While a large percentage of the mortuary material is furnished by tuberculosis of the lung, the statistics show more than an equal number in which the tuberculosis of the lung was completely cured or circumscribed and did not contribute in any way to the death of the patient. So common are the evidences of repaired, cured tuberculosis in the lung that the Germans have an axiom that every man at the end has a little tuberculosis. In 1000 necropsies, excluding 216 cases that died of phthisis, Osler found undoubted evidence of previous tuberculosis in 59 cases, 7,5 per cent., but Bollinger, Massini, and Harris placed it very much higher, ranging from 27 per cent. to 39 per cent., far exceeding those that died of tuberculosis. Bouchard, in his report of postmortems, found that in over 75 per cent. of persons dying suddenly there was some evidence of tuberculous lesions, active or obsolete. A comparison of the postmortem evidences of repair of tuberculosis in the various tissues shows that the lung far exceeds any other tissue in the body, not excepting the peritoneum, in its ability to overcome the effects of tubercular inoculation. This repair is effected by the development, from fixed connective-tissue cells, of resisting capsules of connective tissue with sclerosis and cicatrization of the walls of the cavities. These walls resist the biotic and toxic effect of the bacillus; subsequent degeneration of the encapsulated products takes place, or there is an elimination of the infected focus with its bacilli by necrosis and exfoliation, as illustrated after the use of the Koch tuberculin injections. Investigation shows further that the so-called "deaths from tuberculosis" are rarely due to the tuberculosis itself, but to secondary infections, that is, it is not the tuberculosis which kills but the mixed infection (pus and saprogenic) of the tubercular centers; the same is true of tuberculosis in other tissues. Do postmortem examinations show us how the encapsulation of tuberculosis is favored or accomplished? Yes, to a limited degree. Does clinical experience, surgical or medical, offer any suggestions or proofs? Yes, both furnish many if they are properly interpreted. Tuberculosis of the lung is primarily in the great majority of cases a local disease involving but a small portion of one lobe of the lung. Ewart and Kingston Fowler described this process and showed that its extension followed well-defined routes. It advances from the original pulmonary focus by continuity, through the lymph-spaces and lymphatic channels and through the blood, and rarely, if ever, along the mucous surface on which the myriads of bacilli are transmitted to the mouth. Pathology teaches us that the effect of the biotic and toxic stimulation of the bacillus is to produce lymphoid cells and a multiplication of the fixed and connective-tissue cells of the part; they induce increased deposit of leukocytes and vascular changes, with subsequent necrosis and caseation of the center of the nodule

(Baumgarten). In tissues containing epithelium these cells also proliferate and the connective-tissue cells yield epithelioid and giant cells (Gaule and Arnold). Further, the tendency in most tissues in the body is to wall in the destructive process by the formation of a capsule. Tubercular lesions represent reaction of the tissues to the irritation of the tubercle bacillus. In the center of the foyer the irritation is sufficient to destroy the tissues; farther off the irritated tissues build a wall, which in time makes a cicatricial barrier, plugging up lymphatics and constricting vessels. Leukocytes and wandering cells are attracted by dead bacilli. Living bacilli stimulate connective tissue alone (Evans). We have still further abundant proofs that this capsule is destroyed or lessened by secondary infections; that its limiting power is enhanced and favored by physiologic and anatomic quiescence. Its strength and resistance are further favored by aseptic stimulation of cell-proliferation in its periphery. The study of the literature on repaired tuberculosis of the lung shows that there was an atelectasis and cicatrization of the pulmonary tissue in close proximity to the nodule; that there was a fibrous and cicatricial development often in the entire lobe which was repaired; that the pleura in the neighborhood of repair was greatly thickened, and effectually immobilized the lung contiguous to it; that the chest-wall was contracted and motion of the diseased part was restricted. In other words, the histology of the repair of tuberculosis of the lung in its entire range, from the solitary tubercle to a complete lobe, is made up of a series of defunctionalizing processes, anatomic and physiologic, in every tissue involved directly or indirectly.

Treatment.—Hiller, Berlin, hoped to aid the cicatrization and contraction of the lung by the injection of irritating material into the lung itself, as iodin, iodid of potassium, and carbolic acid. His method was tried by Ewald, Pepper, and Jablovoski; they failed to obtain good results. Lepine injected sublimate solution into pneumonic lungs and observed a rapid diminution of temperature and improvement in breathing. Contraction of the chest-wall for the cure of tuberculosis by means of cauterization of the chest was practised by Guerin and Vidal. They reported

37 good results in 44 cases. Mosler tried this treatment in 20 cases, and while he found great improvement in many he does not report a single cure.* Rive injected iodin and iodid of potassium into the cavities with only temporary relief; he had one fatal result from cerebral embolus.

The pathology of repair of pulmonary tubercular cavities involves certain physical conditions which are peculiar to the chest (they exist to a certain degree in abscess of the bone and brain), namely, the constant resistance of the bony framework against contraction, the effort at expansion of the cavity in each inspiratory act. The best illustrations exist in large empyemic cavities. The bony framework of the chest-wall admits of a certain degree of contraction of the cicatricial tissue which makes up the boundary of the empyemic cavity, and then contraction ceases and the cavity remains permanent. This element was overcome by the operations of Estlander, Schede, Delageniere and their modifications, all based upon the same principle, reducing the bony re-

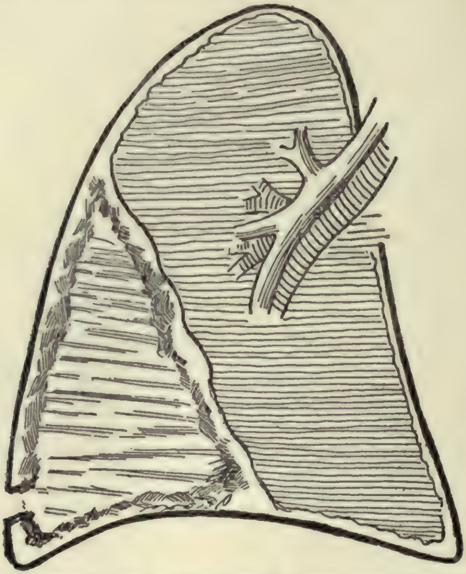

Fig. 235.—Shows empyemic cavity after partial contraction has taken place following drainage.

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* Verhand. des Cong. f. innere Med., Wiesbaden, 1883, vols. i, ii, p. 82.

sistance: all artificial means of allowing the obliteration of a cavity by cicatricial contraction and agglutination of its walls. This same resistance to nature's repair exists in every pathologic pulmonary cavity in addition to the ordinary abscess conditions. Allow the wall of the abscess to collapse, to empty thoroughly, and it will heal as other abscesses of the same pathologic character. This I believe is the keynote to the successful treatment of pulmonary cavities. How to overcome this resistance of the chest-wall was a stimulus to my investigations in lung surgery and led to the hope that not only might pulmonary cavities be obliterated by permitting or forcing their collapse, but that primary tuberculosis might be encapsulated by mechanical immobilization of the diseased portion by collapse and enforced rest of the lung as a respiratory apparatus, thus allowing the cicatrization and encapsulation of the tubercular foci; in other words, that tuberculosis of the lung might be treated as we treat tuberculosis of the joints, by immobilizing and enforcing physiologic rest; by enforcing drainage of the secondary products through the bronchi and allowing the ulcer or cavity to heal.

Are we furnished any evidence from the record of postmortems that may be appropriated to support the principle that immobilization and physiologic rest of the lung are favorable to the healing of pulmonary tuberculosis and pulmonary cavities? Yes. (1) Lungs that have been severely involved in tuberculosis, empyema, pneumonia, gangrene, or abscess when repaired show a contraction of the chest-wall in proportion to the degree of tissue involved; (2) lungs with tuberculosis that have been compressed by pleuritic exudates, usually sequences of the pulmonary tuberculosis, have repaired the primary tubercular focus during their quiescence; (3) lungs that have been allowed to collapse following radical operations for the obliteration of empyemic cavities (tubercular in a large percentage of the cases) have healed.

That empyema is curative of tuberculosis of the lung in its early stage I believe can be readily proved. The great majority of empyemas in the adult is the result of primary pulmonary tuberculosis—83 per cent.; in children tuberculosis is rarely the cause of empyema. In most of the postmortems on chronic purulent tubercular pleurisy tubercular lesions of the lung or other organs are found, but these lesions are generally quiescent and localized. Empyema from tuberculosis never absorbs and rarely opens into a bronchus. It is a result of mixed infection of the primary tubercular nodule in the lung extending to the pleura (Strauss). Of 164 uncomplicated cases of empyema operated by Koenig, Cabot, and Runeberg, 7 remained with fistulæ and were incomplete cures, 4 died, and 9 were lost sight of before the cure was complete; all the others recovered. This shows what a favorable termination empyema may have, and considering that it is secondary to tuberculosis in a great majority of cases (83 per cent.) it speaks forcibly for its curative effect upon the lung. Whitney* says "the course of tubercular empyema is peculiarly benign." In 16 cases of pleuritic, non-suppurative effusion, Netter found bacilli in all. It is the prevalent opinion of pathologists that pleurisy from "cold" rarely if ever occurs. Strauss quotes from German authors that "simple" pleurisy is tubercular pleurisy, the true nature of which is not recognized; tuberculosis is the usual etiologic condition in pleurisy; one can say that it represents the real cause in over three-fourths of the cases. Landouzy regards pleurisy from "cold" as absolutely exceptional. Kelsch and Villard, who investigated 16 cases, say that pleurisy is only a local manifestation of tuberculosis. Chauffard and Gombault inoculated the fluid from 20 simple pleuritic exudates in guinea-pigs; 10 died of tuberculosis, the negative cases do not prove that they were not tubercular. Every general practitioner recalls numerous cases of tuberculosis of the lung resulting in empyema or hydrothorax in which there was a complete recovery of the tuberculosis when the empyema and hydrothorax were operated at the proper time.

When I began this line of investigation I had hoped to find in the reports of the great pathologic institutes detailed accounts of the condition of the primary tubercular focus in the lung and prove my position by them; so far I have been unable to find this material, as the postmortem records at my disposal have been defective in this particular. I have called the attention of a number of pathologists to this point and hope shortly to be able to submit accurate descriptions of the tubercular process in the lung with empyema. I do not believe that the empyema or the hydrothorax is advantageous to the tubercular process by an inhibitory power exercised by the fluid or by a venous stasis, as suggested by Fowler,* of Brooklyn, in his able article, "Surgery of Intrathoracic Tuberculosis." On the contrary, from this standpoint it is detrimental, as it impairs the general condition of the patient. It increases the immediate danger, but favors the ultimate result. Nothnagel and Schrader do not remove the effusions primarily. The force of numbers of operated cases of empyema with permanent cure of the primary tuberculosis of the lung carries conviction that the compression of the lung favors the reparative process, and in this way the pleuritic effusion is curative. Quiescence of the lung greatly diminishes lymphatic circulation and thereby lessens materially the likelihood of the propagation of the tuberculosis to the other parts (W. A. Evans), a fact of great moment, as the tuberculosis is disseminated, from a practical standpoint, almost exclusively by the lymphatics. The methods of obtaining pulmonary quiescence or collapse are: (1) By permitting or forcing the collapse of the lung by separation of the parietal pleura and intrathoracic compression (extrapleural pneumothorax); (2) by removing the bony resistance and allowing the collapse of the chest-wall, thus favoring contraction and cicatrization (thoracoplasty); (3) allowing the lung to collapse by intrapleural injections of gas or fluid. The third method appears to offer more than any of the others. We know that atelectasis of the lung may exist for months, or even years, and the lung be again restored to its function, as shown by West's cases of six to eighteen months' compression with effusion, which was followed by immediate restoration of the lung to full size after removing the fluid; (2) the treatment is simple and painless; (3) the patients will consent when the pathologic condition is most favorable.

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*Twentieth Century Practice of Medicine.

Good results were obtained in tubercular cavities with thoracoplasty by a number of authors. De Cazenville and Sengler report recoveries from disease of right apex. Baier reports a temporary recovery. Stewart Tiday supplemented this operation with external compression with adhesive plaster. Coromilas and Macewen favor thoracoplasty.

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*Ann. of Surgery, 1886, vol. xxiv, p. 591.

Our experience, however, was not satisfactory. Friedrich, of Breslau, has used this quite extensively; what his final conclusions are, I do not know. Too, the surgical attack is too severe on a patient whose resistance is already impaired by disease, and the repair of the disease depends so much upon increasing the resistance and not on depleting it. We therefore early abandoned it as a regular means of treatment and supported intrathoracic compression with nitrogen or sterile air as the ideal method for immobilizing the lung.

I have performed the following operation for the purpose of allowing the lung to collapse over a tubercular cavity:

Case 1.—Stephen D., English, aged forty, single, clerk, Cook County Infirmary, Surgical Ward 7. Family history: Tuberculosis caused death of mother, two sisters and two brothers. Has had tuberculosis for three years; is emaciated and anemic. The upper lobe of right lung is consolidated; has evidence of a large cavity extending from the first rib to the lower margin of the third with a transverse diameter of two inches; has marked cracked-pot sound; he expectorates two ounces of material with each seizure of coughing; râles over the upper portion of the right lower lobe behind and some râles over the upper lobe of the left lung; no evidence of consolidation in this lobe; tubercle bacilli present; patient had an evening temperature ranging from 102° to 102.5° F. It was decided that he was not in a condition to drain the cavity, and a compression of the lung over the position of the cavity was determined upon.

Operation.—January 9, 1898, chloroform narcosis. Immediately before operation the patient's temperature was 100°, pulse 98, respiration 28; there was a frequent hacking cough. A U-shaped incision was made, beginning at the sternum and the second rib, extending down to the third and then three inches to the right and up again to the second rib; the skin and muscular flaps were elevated; subperiosteal resection of three inches of the second rib; the parietal pleura was detached from the chest wall for a considerable area downward and outward, so as to allow of a greater compression of the lung at that point. The flap was replaced and retained with a deep musculocutaneous suture. Very little chloroform was used, as the patient was somewhat cyanotic when the operation began; it was feared he might die on the table. He was placed in bed and rallied rapidly.

[Immediately following this there was a gradual subsidence of the cough and disappearance of the temperature.—Ed.]

From this time on his temperature, pulse, and respiration have been normal; there was primary union of the wound and great depression of the chest over the site of operation. The patient's expectoration has diminished more than half, and is of a light, foamy character. His improvement was progressive. He has gained in weight and appearance. April 30 physical examination reveals an entire absence of râles and bronchial breathing on the right side; there is diminished respiratory sound over the seat of depression of the chest-wall; no physical signs remain to indicate that a cavity had existed in this locality. On the left side there are râles extending down to the nipple. These physical signs were verified by Drs. George W. Johnson and W. A. Evans. The repair of the lung which has taken place can be seen in radiograph of the right lung; no cavity exists. If the lung in this case were involved to a greater degree, I could have inserted my entire hand through the opening in the chest-wall into the chest cavity and separated the lung from its adhesions to the wall and compressed it.

Examination July 8 shows that the tuberculosis in the right lung was entirely quiescent. There was not a râle to be heard; the cavity was obliterated; there were some râles over the upper lobe of the left lung and the upper portion of the lower lobe; the patient has had no fever and has had but little expectoration.

Dr. Alexander C. Wiener and Dr. James G. Berry examined this patient with me.

Carron proposed to compress the lung over tubercular cavities with the hope that the collapse of the lung would be followed by an obliteration of the cavities. Macewen favors resection of the ribs to allow the contraction of the chest over small cavities and the drainage and even curetment of large ones. Coromilas, Greece, favors pneumotomy and early surgical interference in tubercular processes.

If the adhesions be firm and the parietal and visceral layers cannot be separated, then the parietal layer should be separated from its costal attachments and allowed to sink in with the lung, or it should be held compressed by a tampon beneath the flap for forty-eight to seventy-two hours. Will found that in 36 cases of tuberculosis in the early stage, 32 admitted of complete collapse of the lung and 4 only partial. If on examination it be found that the lung is free from adhesions, or that circumscribed adhesions exist, then the treatment is entirely different; the lung may be forced to collapse by the injection of a fluid which is slowly absorbed, as the mineral oils, albolene, etc., or, better still, it may be compressed and retained compressed by the injection of nitrogen gas, after the method described for the determination of pleuritic adhesions. That this method is practicable, as far as its technic is concerned, I have demonstrated in the following cases. It can be given with little more pain than a hypodermic injection. The patients suffering from tuberculosis of the lung on whom I used this treatment are progressing favorably. We have had no postmortems to show the reparative effect upon the tubercular process. The radiographs show distinctly that the lungs remain compressed. The *x*-ray is of inestimable value in studying pathologic processes of the lung, particularly in the localization of cavities. The degree of compression of the lung can be photographed and the rapidity of absorption of gas estimated by comparison of pictures. The outline of adhesions when gas is injected is pictured on the plate. The following histories illustrate the method of injection and its effect:

Case 1.—Charles K., aged twenty-three, Cook County Infirmary, was admitted to the hospital April 30. He had suffered from tuberculosis of the right apex since November, 1897. Physical signs showed that it extended down to the lower margin of the third rib; there was no evidence of a cavity; tubercle bacilli were found in the sputum; patient had been coughing for three months; this was so frequent and persistent that he could not sleep. His temperature on the evening of his admission was 102° F. He had been having night-sweats, was somewhat emaciated, and his appetite was poor. It was decided to immobilize the lung. One hundred and twenty cubic inches of nitrogen gas was injected in the sixth interspace after the method described above. The lung collapsed and he presented all of the signs of pneumothorax. The heart was displaced to the left ⅝ inch. The patient's breathing while on the table was a little rapid and the effort somewhat labored. He was up and about the room in five minutes and stated that he felt no distress whatever from the treatment. His cough was almost immediately relieved; he slept the following night without a coughing spell; during the day there was a superficial coughing effort to "clear the throat," as he expressed it. Eight days after the injection he came to my office and I examined him and found that the pneumothorax still existed. There was a small area of emphysematous crackling in the neighborhood of the puncture; distant tubular breathing could be heard; the patient's temperature had lowered to 99.7° F. in the evening and was usually below normal in the morning. I had a radiograph of his chest made at this time. I have seen this patient every week since and his condition continues good. The last radiograph shows that but a very small quantity of the gas had disappeared, which I believe has escaped into the cellular tissue of the chest-wall, as the area of the emphysema has increased.

Examination July 8: The pneumothorax had not diminished in the least. There was not a respiratory sound on the right half of the chest. The hepatic dulness was down almost to the margin of the ribs; the intercostal spaces bulged; there was a tympanitic percussion-note over the right half of the thorax; the pulmonary quiescence was still complete. The patient had not coughed or expectorated a particle in five weeks; eats and sleeps well. He had had no fever and his temperature at 4.30 today is 98.2° F. He had not been weighed, although he feels that he has gained in weight. It is two months and a half since the injection was made and the quantity of gas is practically the same, showing that the absorption of the nitrogen is extremely slow indeed, if any of it is removed by that process.

Dr. Alexander C. Wiener and Dr. James G. Berry examined this patient with me.

Case 2.—E. L., male, aged thirty-four, Cook County Infirmary, was in the second stage of phthisis, with cavity in right upper lobe. Endeavor was made to fill the right pleural cavity with gas, but as adhesions were found it could not be accomplished. He was not in condition to withstand operation for compression of the chest-wall. The gas entered the cellular tissue of the chest and remained there three weeks, showing how slowly it is absorbed.

Case 3.—H. A. E., male, aged thirty, Alexian Brothers' Hospital, admitted May 10, 1898. Family history shows several cases of tuberculosis. His cough and fever began about October 1, 1897. Cough very severe, expectoration limited. Night-sweats since January, 1898. There were physical signs of apical tuberculosis on the right side; patient gave no history of preexisting pleurisy; he was placed on the table, trocar inserted in the sixth intercostal space, anterior axillary line, and two hundred cubic inches of nitrogen gas was injected after method described above. It was found when the trocar entered the pleura that it required no pressure to force the gas into the pleural cavity. It produced very slight coughing and but little anxiety to the patient from shortness of breath; the pulse was increased in frequency to 96 and the lips were discolored a light blue; the apex-beat was displaced one inch to the left, the hepatic dulness three inches downward; the respiratory sounds entirely disappeared on the right side; respirations were increased to 26. Patient was removed to his bed; thirty minutes later was sitting up in bed eating his dinner; he said "he felt a binding in the chest and a tickling sensation in the throat"; he remained in bed for two days, as his respirations were a little increased in frequency and he complained of some discomfort in the episternal region. The radiograph shows the compression of the right lung. Examined May 20. The respiratory sounds still absent; the cardiac displacement continues; there is still absence of the hepatic chest dulness; his respirations are normal and his cough has disappeared; no expectoration.

June 30 patient presented himself again for examination; he thought his breathing was still a little short. Cough has entirely ceased; he has had no night-sweats; has gained 10½ pounds in three weeks; his appetite is excellent.

Physical Examination.—The respiratory sounds are returning, though still distant; there are no râles; the percussion-note shows that the chest has still a considerable quantity of gas, the liver dulness is absent down to the margin of the eighth rib, and the apex-beat is in about the normal position. The mediastinal dulness is to the left of the margin of the sternum; there is some bulging of the intercostal spaces, though not so marked as in previous examinations.

Case 4.—P. C., male, aged twenty-three. Admitted to Alexian Brothers' Hospital six weeks ago. Patient was in second stage of tuberculosis of the right lung. May 12 an effort was made to inject the gas in the usual manner, but adhesions prevented the admission of the gas.

Case 5.—Alexian Brothers' Hospital, E. D., May 12. Patient in first stage of tuberculosis in the right apex; has had cough, expectoration, night-sweats, and fever since August, 1897; bacilli in the sputum; had frequent attacks of pleurisy. Method of injection of gas the same as in Case 1. Puncture made in sixth intercostal space, midaxillary line. When parietal pleura was penetrated the gas readily passed into pleural cavity; seventy cubic inches were injected. Physical signs of pneumothorax rapidly appeared; liver dulness displaced downward one and one-half inches; apex-beat displaced one-half inch to the left. Patient said he felt "his breath was shorter." The opening was sealed. In one-half hour the patient said he felt no inconvenience whatever from the injection, and was walking about the ward. He left the hospital a few days following his injection on May 12. I did not see him again until he presented himself for examination June 30. He was greatly improved in appearance; there was a slight cough, but no expectoration; he had gained in weight; his appetite was excellent.

Physical examination showed that there had been some absorption of gas, although a tympanitic sound was distinctly present over the former area of liver dulness. The respiratory sounds were feeble; there were no râles. Seventy cubic inches of gas was injected in the usual manner. It did not produce the slightest discomfort. After the injection the liver dulness was displaced almost to the costal arch. There was no displacement of the heart. There was some bulging of the intercostal spaces. Patient left the hospital immediately for his home.

July 7, at Alexian Brothers' Hospital, two cases of apical tuberculosis were injected; in one 75 and in the other 80 cubic inches of gas was used; in neither was there the least discomfort from the insertion of gas; in a third case adhesions were found, making it impossible to succeed in the injection. The cases in which I believe this method most practicable are apical or monolobar tuberculosis in the early stage, as the pathologic conditions are such that the compression of the lung can be accomplished and adhesions are not likely to be found. I do not consider that it is indicated nor practical in advanced or chronic tuberculosis, as the fibrous tissue deposited in the lung will not permit compression of the lung, nor will the pleuritic adhesions allow of gas injection. So far no conclusion can be drawn as to the ultimate result of this treatment. Theoretically its dangers may be

—(*a*) hemorrhage from wounding the intercostal vessels; (*b*) injecting gas into the intercostal vein, the possibility of which I question; (*c*) infection by the use of impure gas, infected trocar, or improper antiseptic preparation of chest; (*d*) rupture of infected foci into the pleural cavity through compression of lung and separation of pleuritic adhesions; (*e*) dyspnea from the use of too large quantity of gas; this can be readily relieved by changing the direction of the current in the siphon; (*f*) puncture of the lung with the cannula. This will not take place unless adhesions are present, and if the lung should be punctured it is a matter of no consequence, as experiments and clinical experience show that the lung can suffer great traumatism without danger. While these possibilities of danger are mentioned, none of them occurred in my experiments. I believe the gas should be reinjected every six to ten weeks, as might be deemed necessary in the individual case, depending upon the amount of absorption. If the respiratory sounds return the injection should be repeated. So far I have repeated the injection in but one case.

[This was written in 1898, and shows that the position taken by the profession at the present time is in consonance with the principles laid down by us at that time.—J. B. M.]

Dr. A. F. Lemke at that time also read in the Section on Practice of Medicine, at the meeting of the American Medical Association at Columbus, in June, 1899, a paper entitled "Report of Cases of Pulmonary Tuberculosis Treated with Intrapleural Injections of Nitrogen, with a Consideration of the Pathology of Compression of a Tuberculous Lung." It was printed in the Journal of the American Medical Association on October 14, 21, 28, 1899.

BONE CYST OF THE RADIUS

HISTORY

PATIENT, female, aged twenty-five, enters hospital complaining of enlargement above and limited motion in left wrist, with a discharging sinus in the center of an old scar on the back of the wrist.

In 1905 she fell on a street-car platform with books under her arm, striking on the back of the left hand with the hand flexed. When she got up she could not move the wrist. The skin was not broken. She consulted a doctor, who said she had a Colles' fracture, and set it one-half hour later by pressing on the extensor surface of the forearm with his thumb. He applied a straight splint on the flexor surface extending from the elbow to the finger-tips. The splint was removed in two weeks and a cast was applied to the whole forearm, with the hand midway between flexion and extension. Four weeks later the cast was removed. At this time she had very little motion in the wrist and some pain on motion. There was a swelling on the back of the wrist. Two weeks later an adhesive tape cast was applied and left on two weeks. Motion did not improve any. The doctor made a skiagram and said she had tuberculosis of the bone. The forearm was put in a cast for five months, during which time she took *x*-ray treatments and the back of wrist was burned. The doctor aspirated the tumor about this time, and said there was nothing in it except a bloody fluid. Since the accident she has been able to flex wrist but little, and she has pain in the forearm whenever she attempts to use the wrist much or when she lifts objects with that hand; also when the weather gets cold and damp. Sometimes the pain extends to the shoulder. From 1907 to 1911 she had enlarged glands in both axillæ, but especially the left, where the glands were opened twice and discharged pus.

She does not know whether she had any temperature elevation at this time. The left wrist is perceptibly larger than the right. She was always troubled with tonsillitis until tonsils were removed four years ago. When fourteen years old she had scarlet fever, followed by a bad attack of acute nephritis, with blood in the urine and convulsions She was sick about eight weeks. An uncle and aunt died of pulmonary tuberculosis. Father has diabetes. Mother living and well. One sister and one brother died in infancy. No Bence-Jones bodies in the urine.

Examination.—The skiagram shows an enlargement of the lower end of the radius, with an absorption of the adjacent surface of the ulna. The enlargement is pyramidal in shape, regular in outline, and of a uniform density, except on the edges. These are much more dense, evidently because of their consisting of compact bone. The periosteum appears to be thickened. The neighboring structures are not infiltrated. It is undoubtedly a bone cyst or a fibrosis preceding cystic degeneration. The carpal bones are not very clear cut. If they are diseased, this is a tuberculosis, which we do not believe to be the case.

COMMENTS AND OPERATION

DR. MURPHY (Operation June 30, 1913): How old is she?

INTERN: Twenty-five.

DR. MURPHY: How old was she at the time of the injury?

INTERN: Seventeen.

DR. MURPHY: How long after the accident was that bone punctured?

INTERN: About seven months.

DR. MURPHY: And the doctor aspirated a bloody fluid. Is this a tuberculosis involving the carpal bones, which the skiagram might be interpreted as showing, or is it a bone cyst? If that was a bloody fluid in there, there is no question but that it is a cyst. The skiagram is not clear. It merely shows that all the carpal bones are separated, free, and independent. The carpal bones should not be involved if this is a cyst, and they might be if it is a tuberculosis. If this were a tuberculosis, beginning at seventeen, it should have commenced in the epiphysis. It appears

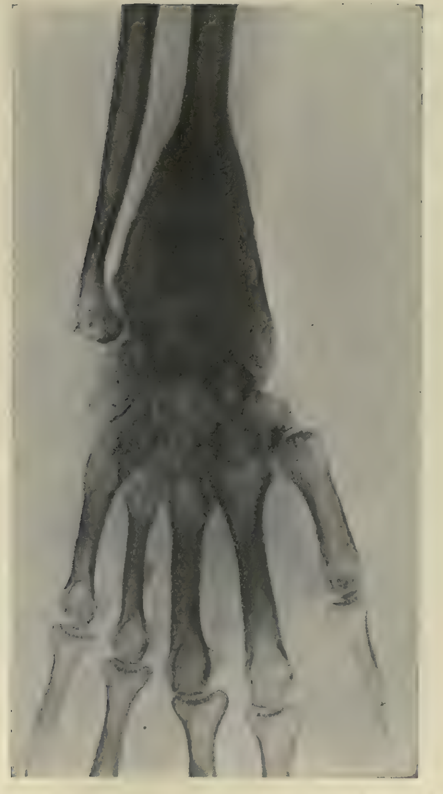
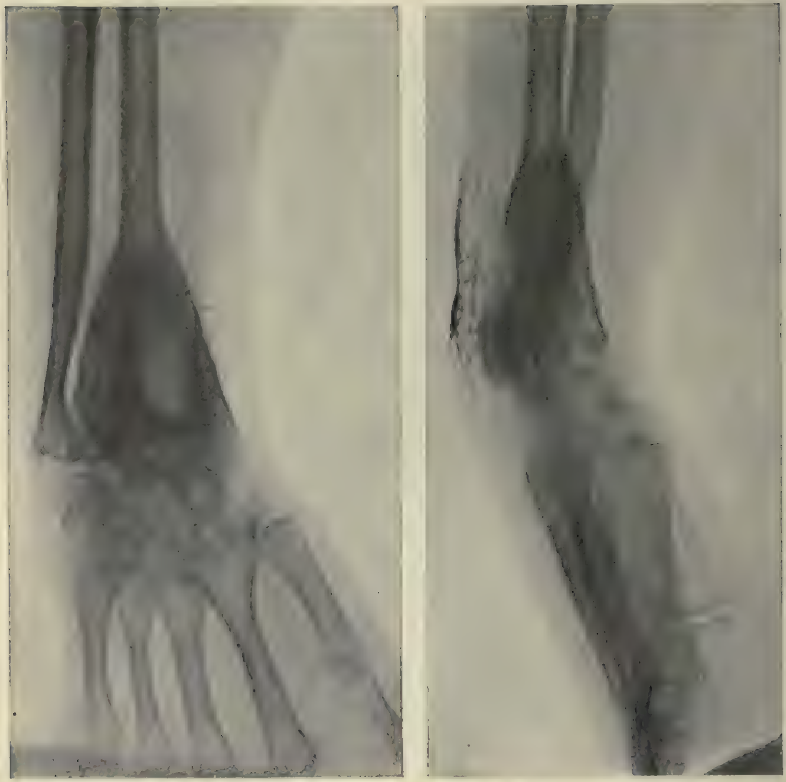


Fig. 236.—Tumor of the radius. Anteroposterior view of bone cyst in lower end of radius. The tumor has encroached on the ulna, producing an absorption of the bone. The outlines of the cyst are definite and regular, showing no involvement of the surrounding tissues. There is no ankylosis at the wrist-joint.



Figs. 237 and 238.—View of same case. Anteroposterior and lateral views taken about two weeks after operation. There does not appear to be any change in the picture, except that in the center of the mass in the anteroposterior view is a lighter shadow, marking the spot where the roof of the tumor was broken down to allow of the curetage which was performed in this case.

as though this had been a primary lesion of the shaft and later involved the epiphysis. Tuberculosis in adults always begins in the epiphysis. Tuberculosis in infancy and childhood occasionally begins in the metaphysis. Cysts never begin in the epiphysis—they always begin in the metaphysis. This girl is twenty-five years old, and there is no line of demarcation of the epiphysis at all, meaning that the lesion, if it is a tuberculosis, began where tuberculosis rarely ever begins at that age, namely, in the metaphysis, and that it extended, involving not only the epiphysis, but the upper row of the carpus. Tuberculosis is essentially an epiphyseal disease, but in Scotland, in Mr. Stiles' clinic, they occasionally have tuberculosis in the diaphysis in children five, six, and seven years of age, which we practically never see. Whether that is due to the feeding of so much milk, with a possible bovine tuberculosis, focalizing differently, which causes the disease in the diaphysis, which we do not have, I do not know. I have seen only one case of tuberculosis in the diaphysis in infancy. In infants it is an entirely different proposition, because in the infant the epiphysis is cartilaginous and you never have a primary infective lesion in the cartilage.

There is no ankylosis of the wrist. Examining the skiagram, one would think that there was an ankylosis, but that overlapping of these bones is due to the deformity that exists here from the habit of holding the hand up in moderate extension. When the *x*-rays went through, they did not go through in the right direction to show this articulation. There is motion in the wrist—rather a free motion. The discharge comes from this sinus on the back of the forearm over the tumor, and was caused by the *x*-ray burn. She was given *x*-ray treatments for a while. We believe that this is a bone cyst. We have had two of these cases in the past few months. [See August CLINICS.]

After the usual preparation of the skin, painting it with a 3.5 per cent. tincture of iodin, we make an incision on the back of the arm, two inches in length, excising the old scar. Retracting the edges of the incision, the tumor is easily exposed. The extensor tendons have been displaced to both sides by the tumor. The periosteum is not materially thickened, showing that there is no acute inflammatory process in this neighborhood at this time. We split the periosteum and push it back on both sides with the periosteotome; then we break down the canopy of this tumor. Granulation tissue is filling this cavity. That does not look at all like a tubercular "brie"; it looks more like the tissue of an advanced myeloma. The tumor has a clean-cut margin. Its inner walls are hard, compact bone. The bones of the carpus show no perceptible enlargement, and they are normal in every way. This looks more like a myeloma than anything else. We will now fracture the internal and posterior wall of this tumor in many places, and depress it into the cavity to hasten the subsequent ossification in the dead space left after curetment. This, you will see, is easily accomplished.

Let the record show that the cavity is filled with granulation tissue; that its walls are hard and firm; that they consist of compact bony tissue, showing that there has been a constant irritation, which has caused this cicatricial bone. The tumor is about as large as a walnut, and is filled with granulation tissue. We cureted this thoroughly, removed all the granulation tissue, fractured and depressed its walls, and let the periosteum come together over it. We do not need to fill this cavity with a transplant, bone-chips, or any other material, because of its small size. The skin wound will be closed with horsehair.

[Stitches were removed on the tenth day. Primary wound healing. Patient left hospital two weeks after the operation. —Ed.]

PYONEPHROSIS: DRAINAGE

HISTORY

Patient, female, aged thirty-five, enters hospital because of a mass in the left side of the abdomen.

Six or seven years ago she began to have attacks of colicky pain in the left lumbar region. The pain persisted for three to eight hours, was of a colicky nature, and she had to lie down until it stopped. Pain always began when she was in a stooping posture. Attacks came on at intervals varying from one week to three months, depending on whether she was doing work which necessitated stooping or lifting. After three or four years these colicky attacks ceased, but she has had tenderness and some pain in the left lumbar region ever since. During the past four months the pain has been increasing in severity and is becoming more constant, radiating down the back of the left leg to the knee. The left lumbar region feels tender and "aches." About four months ago she noticed several times that the urine was blood-tinged. Has not noticed any blood in urine since. Patient can feel a mass in the left side of the abdomen the size of a grapefruit, freely movable, hard, and extremely tender. She says it seems to float in the abdomen; that it changes position when she does.

No menstrual disturbances. August 13, 1902, a myomectomy was performed for fibroid. April 15, 1903, she had another operation—anterior fixation of uterus by shortening of round ligaments. She made a complete recovery from both operations.

The urinalysis made this morning showed a specific gravity of 1013, acid reaction, no albumin, sugar, or casts, many epithelial cells, and three pus-cells to the one-sixth field. She has a leukocyte count of 8400.

COMMENTS AND OPERATION

Dr. Murphy (Operation July 2, 1913): This patient has been operated on twice before. First she had a uterine fibroma, which was removed by a myomectomy. Then the uterus retroverted into the Douglas pouch, for which we did an anterior fixation of the uterus and a shortening of the round ligaments. Four years later, in 1907, she began to have attacks of pain in the left side. Did you say it was recurrent, doctor?

Intern: Yes.

Dr. Murphy: I did not get that interpretation of it. My interpretation of it was that the pain would get quite severe, but not of the wavy type, and that when she would lie down flat on her back it would be relieved—just the type of symptoms that one would have in these cases. She had no nausea and vomiting. She did not have a typical Dietl's crisis. That occurs with sudden severe pain, nausea and vomiting, sensitiveness in the side, and is relieved suddenly by change of position, which means that it is a luxation of the kidney with obstruction to the outlet. I believe that that obstruction occurs not only to the outlet, namely, to the ureter, but that it is also an obstruction to the circulation. The symptoms continue to recur in that way with shorter intervals. That history would lead one to say that this was a displacement pain. The condition went along until five months ago, when she began to have symptoms of hemorrhage. What was the type of blood that was present?

Intern: The urine was just blood-tinged.

Dr. Murphy: What else? What else should be in the history? "Angle-worm" formations should be mentioned, because when blood comes from the kidney from rapid hemorrhage, there should be "angle-worm" formations. That would be conclusive evidence that the blood came from the kidney, because with the other symptoms it might not. The blood may come from the ureter or from the bladder, as when it is the seat of a papilloma. There are many sources of blood in hematuria which can be differentiated only through the careful macroscopic examination of the urine voided in separate receptacles.

Considering the symptoms present in this case, what is the diagnosis of the pathologic condition of this kidney? In about 70 per cent. of the tumors of the kidney the first symptom is hemorrhage, not pain. The hemorrhage may be present for a long period of time before the pain occurs. The next symptom in frequency is pain. The third symptom is nausea and vomiting associated with the pain, and fourth, the presence of the tumor. That is the order of occurrence of the symptoms. In this case the order was reversed. The pain came first—years before the hemorrhage appeared. If the hemorrhage came first, then you would say at once that it was probably one of the malignant tumors, because that is the type of tumor that almost always gives a history of hemorrhage as an early symptom, or a stone eroding a vessel or a renal tuberculosis. Then consider the years that she had the repeated injuries, which means that the displacement may have been the cause of the hemorrhage. An accessory artery may pass across the pelvis of the kidney or ureter, as described by Israel many years ago, without causing pain. This causes a displacement of the kidney with compression of the ureter and a retention in the renal pelvis, with possibly an obstruction to the circulation of the kidney. Where there is a second artery coming in and crossing the kidney it obstructs the ureter, when the patient gets a luxation of the kidney.

Next the question comes up is this a cystic condition of the kidney,—a congenital cyst, multiple or single,—although this condition rarely gives a history of hemorrhage as a symptom.

TUMORS OF THE KIDNEY

Morris, in 154 cases found:

Sarcoma in 63 (hypernephroma)—17 per cent. (Albarran).
Carcinoma in 41.
Cystic degeneration in 21.
Hydatid cysts in 11.
Adenoma in 10.
Papilloma in 3.
Myxoma in 2.

Lipoma in 2.
Dermoid in 1.

Tumors of the kidney represent 2 per cent. of the total tumors of the body.

Secondary kidney metastases: Of 69 carcinomata, 10 showed secondary kidney tumors.

Rohrer found that one-third of all renal tumors occurred in children.

Kilzneck found that 52 per cent. occurred before the age of ten years. Large proportion before the age of five years.

Mortality from nephrectomy ranges from 25 per cent. (Kuester) to 32 per cent. (Selmieder). In the decad 1880–90, 64.5 per cent. to 22 per cent.; from 1900–10 (Israel), 20 per cent. Percentage of permanent cures for malignant diseases has been accurately estimated.

For your convenience I have had written on the board a list of the tumors that occur in the kidney and the facts concerning them. Sarcoma is the most common,—63 per cent.,—and of this number 17 per cent. were hypernephromata, which are the least malignant of this class of tumors. Carcinoma is the next most frequent tumor of the kidney—41 per cent.; then the cystic degeneration, the hydatid cysts, the papillomata, myxomata, etc. The dermoids, lipomata, and myxomata rarely bleed. The papillomata bleed frequently. The adenomata bleed in only a small percentage of the cases. The hydatid cysts rarely bleed, except when they are ruptured. Of the cystic degeneration, only a small percentage bleed, while in sarcoma and carcinoma hemorrhage is an early manifestation in a very large percentage of the cases. Hemorrhage from the kidney, unassociated or unaccompanied by colic, always demands most serious consideration. Occasionally hemorrhage and colic are caused by blood-clots in the renal pelvis or in the ureter. The passage of the clots causes a colic, so that a colic associated with hemorrhage must not always be interpreted as due to a stone, although in quite a number of instances stones are the common accompaniment, but one is in danger of thus interpreting the symptom because of the severe colic.

In case of neoplasm, the length of time that may elapse between the primary appearance of the tumor and the manifestations of its presence in the way of blood in the urine is very great, that is, the hemorrhage may be a very late manifestation of a tumor that has been present for a number of months, often for a number of years. For instance, take this case: The tumor was discovered early. We interpreted it, first, that this woman has this tumor about six years, if the tumor was present in the beginning; second, the tumor was associated with displacement of the kidney (six years ago). Why? Because it is six years since she began to have colic and pain, but she had no hemorrhage, no blood in the urine, until five and one-half years after the discovery of the tumor, so that the period that elapsed between the primary manifestations and the secondary manifestations is often very great, showing that these tumors may grow slowly and that they destroy life slowly. That is true of the majority of the sarcomata, and practically true of all the hypernephromata. The hypernephromata may be present six, eight, or ten years. That is a very important fact in this case, because you must determine whether or not you will do a nephrectomy. Why? Because a nephrectomy in a case of kidney tumor has a colossal mortality. Israel, who got the best results up to date in a large number of tumors, and who wrote the best book on tumors, because it is a clinical citation of cases, which is the proper way, I believe, to write a book, had a mortality of 20 per cent. in the decad between 1900 and 1910. How much hazard are we justified in taking with these patients by way of operation? Will you take out that tumor or will you leave it? These patients often live so long with the tumor that in only a certain number are you justified in taking out the kidney, because only such a small percentage of sarcomata are permanently cured after you do a nephrectomy and you may immediately shorten the life of the patient.

We will expose this kidney through the lumbar route. The patient is placed on the sound side, with the thighs and knees flexed and a sand-bag under the loin.

There is the anterosuperior iliac spine. There is the tumor, you see. It fills that space between the iliac crest and the ribs. I will carry the incision far forward to the margin of the rectus to get a good exposure, if I decide finally to take out the tumor. We expose the kidney by the usual oblique incision.

There is the iliohypogastric nerve. I want to show you that nerve, because you must never include it in your suture and never cut it. If you do, it gives the patient great pain or a disagreeable anesthesia. The kidney shows now in the bottom of this wound. In cutting the capsule I cut it far behind, so as to avoid opening the peritoneal cavity, because the peritoneum often passes clear behind here in these multiple infections. When you look at this mass of tissue presenting in the wound it looks as though it was peritoneal fat. It is the perirenal fat. This tissue appears edematous. See it there! myxomatous, as it appears on the surface there. I am trying to get an estimate of the character of the mass. It looks like a sarcoma undergoing cystic degeneration. It fluctuates. We will aspirate this mass and see if it is an infection, one of the two things that may have caused that bleeding. You cannot tell the difference between the edema in the margin of a sarcoma when you first inspect it and an infection. [Aspirates and pus appears in syringe.] See what experience does for us! Just look at that! The analysis of that urine did not show any pus present.

Of course we will not attempt to take out this kidney now. What will we do? Drain it. I want you to hear the report on that urine. This is a beautiful case in connection with the findings, which brings out the element we tried to accentuate, namely, whether this was a primary lesion or whether it was a secondary lesion.

Intern: The urine contained three white cells to a one-sixth field.

Dr. Murphy: Three white cells to a one-sixth field; that is less than in the average normal urine, showing that this lesion does not communicate with the pelvis of the kidney. It is entirely confined to the medulla of the kidney. That explains why she went so long without having a hemorrhage—five and one-half years. It is not a sarcoma. That also accounts for the infiltration of the tissue that looked so suspicious of sarcoma —when I said you cannot differentiate between a para-edema of sarcoma and an infection. I will see if there are any other pus-pockets here. To take out that kidney now would materially hazard her life. To do what we are doing has no hazard to life at all, and involves practically no risk. I am not anxious about that pus. Why? Because it is sterile pus, the same as that found in the Fallopian tubes. It does not communicate with the kidney pelvis. It was probably the luxation of the tumor that precipitated the infection of the renal tissues, just the same as an infection may be precipitated in the knee or in the hip. There are two pus-pockets here, one above and one below. We will drain both.

Let the record show that on examining this zone the tissue about it was edematous, myxomatous, which is always suspicious of a rapidly growing sarcoma or an infection. This type of edema is seen occasionally where you have the appendix adherent to the anterior abdominal wall in acute infections. It is not common in the more chronic or resisted infections. You rarely find it close to a cold abscess. That was one of the things that attracted my attention as soon as I got down into the neighborhood of the abscess.

Let the record show further that the larger tube is in the lower pus-cavity and the smaller tube is in the upper cavity.

Let the record also show that the neoplasm fluctuated; that the upper kidney zone was aspirated, and that it contained a yellow, very creamy pus; that the pus was confined to two separate pockets, one in the upper pole of the kidney, the other in the lower; that neither communicated with the renal pelvis; that these pockets were opened and drained; that the remaining portion of the kidney was palpated, but showed no additional abscesses; that two rubber drainage-tubes were inserted. The wound will be closed in the usual way by figure-of-8 silkworm-gut sutures and horsehair for the skin.

The percentage mortality for a primary removal of a kidney suppurating in this way and the secondary removal were first brought out by Morris many years ago—twenty or possibly twenty-five years ago. He showed that a primary removal of a kidney for suppuration had a mortality of something like 33 per cent., while the secondary removal had a mortality of 2.5 per cent., showing the great contrast between the effect of primary and secondary removal.

We will let this drain for a long period of time,—two or three or four months,—depending on how the patient is getting on. At the end of that time we will do a nephrectomy, or an extirpation of the suppurating sacs because, as they are probably epithelial lined, having been primarily retention cysts which became infected, they will not heal by coalescence of the walls, just the same as we managed a similar case some three weeks ago. The patient was here in February with an enormous abscess in her kidney which communicated with the renal pelvis. We put in a drainage-tube, as I did today. When she came back the tissues had contracted down to a cicatricial mass, with very little urine coming from that kidney. The other kidney had taken up the work of both kidneys. Then we decorticated the renal tissue that was left and removed it by clamping the vessels. You know what it means to take out the true capsule with the kidney down to the pelvis, taking out all the pockets and the remaining renal tissue, that had all contracted down into a small mass. We prefer to work inside the capsule. If you split the true capsule, which is always thickened, and get inside of it, then you have left just renal tissue. You know where you are. You have nothing inside but the cortical portion of the kidney—what is left of the cicatricial capsule to the pelvis. There is some oozing from the capsule, but generally very little. Free that kidney all around until you get it up into the field. You must see the vessels at the hilum because they are outside the true capsule. Then we clamp the pedicle with two pairs of rectangular forceps. These forceps have detachable handles, so we just clamp them down on the vessels. Then detach the handles, and tie a gauze strip on the forceps to keep them locked.

At the end of seventy-two hours put on the handles, remove the binding gauze, and detach the forceps. That is all there is to it. The forceps act like an angiotribe. They produce a pressure necrosis. You can see what a comparatively simple operation that is. There is no hemorrhage whatever from the stump. That is not so hazardous as the primary operation, because the mortality is only 2.5 per cent. Primary removal of the kidney is one of the major operations in the abdomen that carries the highest mortality. That is why I have had these statistics put on the board, to give you some idea of what can be expected.

The next question is the permanency of cure. At one time I made this statement, based on the statistics of that period, about ten years ago, that there was no authentically recorded case of true *sarcoma* in the kidney that had survived beyond two or three years without a return of the disease either *in loco* or in the other kidney. If that statement is still true, no one should remove a sarcomatous kidney, because the patients will average more than that period of life if they are let alone, not to speak of the immediate mortality that occurs with its removal, running as high as 33 per cent., Israel giving a 20 per cent. mortality. In the decad between 1880 and 1890 there was a 64.5 per cent. mortality. Removal of a kidney that is secreting nearly a normal quantity of urine with pus, or with a neoplasm in it, is an extremely hazardous procedure. The removal of a kidney that has suppurated and has been drained and that has little or no secreting tissue in it is one of the safest operations, giving only 2.5 per cent. mortality. A rule may be laid down that the nearer the kidney is to performing its normal function, the greater the danger of nephrectomy.

In operating on the left side, where a large tumor must be removed, one must bear in mind that occasionally the left pleura comes 1 or 1½ inches below the last rib, and that one may open the pleura and not the kidney. If you do, all you have to bear in mind is to put your finger on the hole, temporarily plugging it, and then sew it up—sew the muscles around it completely. If much air has been admitted after the operation has been completed, put the needle in the side of the pleura and pump out the air, reëxpanding the lung, just reversing the order of what you do when you have a hemorrhage of the lung. When you have an acute hemorrhage of the lung, one of the first things to do after you give a primary dose of morphin, which is always the first thing to do, is to take a large exploring hypodermic needle and insert it on the side where you think the bleeding is coming from—you can usually tell. Do not use the ordinary sharp needle; grind it off to make it blunt, so that when you come to the visceral pleura, it will not go through the tissue, but carry it in front of it. Cover the butt end of the needle with cotton to form a filter for the air; put your finger on this to act as a valve; elevate the finger when the patient inspires and take it off when the patient expires. By thus alternating you can pump the lung full of filtered air and thus collapse and compress it to any desired degree. This, "the Murphy procedure," we have used with good results for fifteen years, and still the general profession has not seemed to grasp its simplicity, availability, and effectiveness. You do not need any other instruments than a needle, some cotton to filter the air, and your finger to act as a valve. With that process you can press the lung down flat. We did this for hemorrhage in 1898, when we were compressing the lung with nitrogen as a treatment for tuberculosis. The method received little attention in this country, but it was taken up all over Europe, and after receiving a splendid German baptism it is being done in this country now. The other day I received an index of literature references of 384 articles, recorded in the European literature, on compression of the lung for the treatment of tuberculosis. That was the subject of my address in Surgery before the American Medical Association in Denver in 1898. I never dared to do this work after I read the article, because its practice would have taken me out of surgery into medicine. Dr. A. Lemke, one of my former associates, made 1600 to 1700 injections of nitrogen before his death. When he died there was no one to take up this work. In this large number he had only one accident—a cerebral air embolism, which gave some cerebral disturbances for some time after. I now frequently see cases, ten to fifteen years afterward, permanently cured by him.

It is very gratifying to hear that it is coming into vogue again, coming back to this country from Europe, because it is founded on the right principles. Absolute rest is the best treatment for the repair of tuberculosis in any part of the body.

Let the record show that Sister reports the sponges counted and correct.

[The pus was being discharged freely through the drainage-tubes. The patient left the hospital on the twenty-first day. There was still some discharge from the tubes, but the patient was feeling very well.—Ed.]

EXOSTOSIS OF RADIUS AND ULNA

HISTORY

Patient, female, aged twenty-six, enters hospital on account of limited extension and flexion of left arm at elbow. Trouble dates back to when the patient was five years old (1892), when she fell into a hole six feet deep and struck on the point of her left elbow, sustaining a fracture. The arm was put in a cast. When the cast was removed, there was some limitation of motion, but she could bring her fingers to her mouth.

When eight years old she again fell on the elbow and sustained another fracture. The arm was dressed in an extended position; no cast was applied. From that time on motion was more limited, and it has been getting more limited all the time. In the past eight or ten years she has been unable to get her fingers to her mouth. She has had pain in the elbow at times for years, especially during rainy weather. For the past two months she has had a constant dull pain in the elbow, although it has not kept her awake at night. During the past two weeks the pain has been increasing in severity. She has a callus at the upper end of the radius and an elongation of the coronoid process as shown in the *x*-ray. This, she says, has increased in size in the past two months.

COMMENTS AND OPERATION

Dr. Murphy (May 10, 1913): When this girl was five years old she fell, landing on her elbow. She said it was fractured. She was treated for a fracture, with a perfect result. When she was eight years old she fell again, and the diagnosis was made of a second fracture. Following that fracture there was a limitation of motion, but she had fairly good motion until nine months ago, when she began to have gradual decrease of motion, with pain in the elbow and an enlargement over the head of the radius.

If you will look at the skiagram, you will see what is the matter. There is a limitation of flexion because there is an anterior luxation of the head of the radius, so that it strikes the anterior surface of the shaft of the humerus. There is apparently an elongation of the coronoid process of the ulna. Possibly when I remove the radial head the motion in the elbow will be good. The head of the radius is not in the lesser sigmoid cavity. It has become luxated forward, and it has also enlarged. In operating on the anterior surface of the elbow one must avoid injuring two nerves—the ulnar and the musculospiral.

I will have to keep away from these nerves. I will not have great difficulty in getting the head of the radius out. It must be removed. Why? Because it is limiting flexion by impinging on the humerus. We will put a constrictor on the arm. We split a large-caliber rubber drainage-tube, wrap it tightly around the arm twice, with a folded towel underneath, and hold it there with an eight-inch clamp. Why do I want a constrictor? That gives us a complete hemostasis without danger of injuring the vessels or nerves, as I want to see the fibers of these nerves as much as possible in a dry field. We will make the incision on the anterior surface of the arm, just over the head of the radius. The incision will be two inches in length. By retracting the wound-edges, the head of the radius comes into view as it rests above the joint-capsule. There is the musculospiral nerve. By making that field so absolutely dry, as we did by putting on the constrictor, I can see the nerve readily. This is no small nerve—it is rather a good-sized one. I will displace it to the inner side. There is the capsule of the elbow-joint. We will split the capsule so as to reach the head of the radius. There is the head, you see, now nicely exposed. By flexing the arm we feel a gradual muscular resistance, and every time we reach a certain degree of flexion the radius head strikes the shaft of the humerus and the arm cannot be flexed any further.

Testing the musculospiral nerve with the excitor, we find that it responds promptly; showing that the nerve has not been

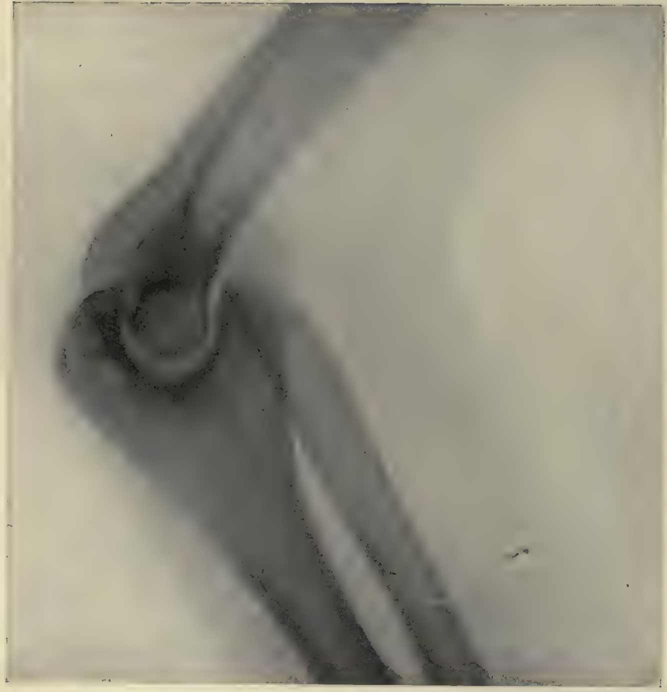


Fig. 239.—Skiagram of elbow made before operation. There is considerable enlargement of the coronoid process of the ulna and some enlargement at the olecranon process. The upper extremity of the radius is lengthened upward a considerable degree, so that on flexion the head of the bone impinged against the anterior surface of the humerus.

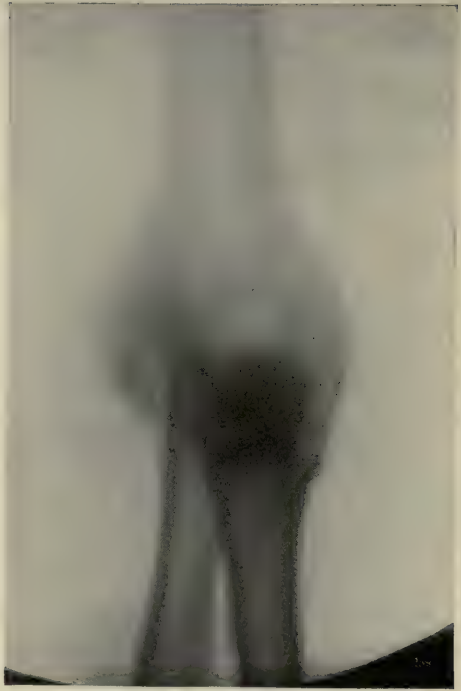


Fig. 240.—Anteroposterior view of same case with arm in extension. The malposition of the head of the radius is apparent. In this picture the radius seems to be about 1¼ inches longer than normal.

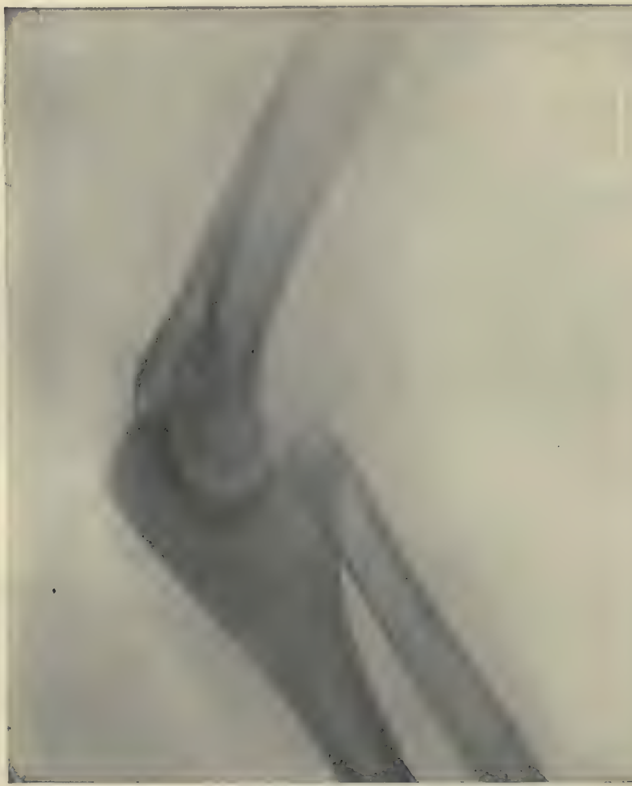


Fig. 241.—Lateral view of the elbow made after second operation. The head of the radius was removed, likewise the coronoid process of the ulna.

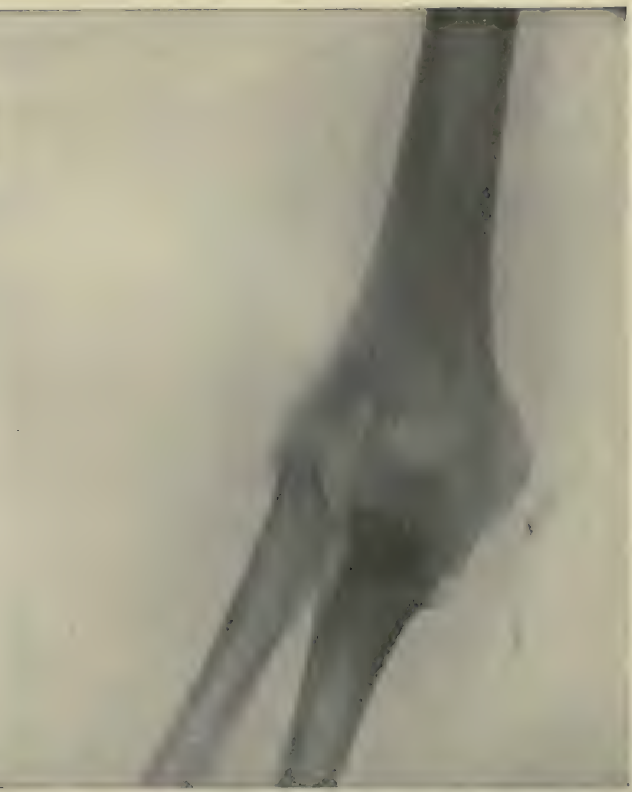


Fig. 242.—Anteroposterior view of same elbow after second operation. The radius now articulates with the lower surface of the external tuberosity of the humerus.

disturbed. I am not going to take off the tip of the coronoid process of the ulna, because I do not want to have two raw bony surfaces in contact or even close proximity as a transverse union might result. It may not be necessary, after removal of the head of the radius. If the coronoid process prevents flexion subsequently, we will remove it at a second operation. The head of the radius is removed with a half-inch straight carpenter's chisel, making the division just above the annular ligament. That was easy. If I had not exposed the nerve, I would have been uncomfortable all the time.

Let the record show that the head of the radius was exposed, and in the dissection we came onto the musculospiral nerve and then its subdivision; that the elongated capsule was then exposed, the head of the radius divided and turned out; that the capsular ligament was left undisturbed; that all the extensor group of muscles at once responded to the irritation of the faradic current; that two small ligatures were placed, and the wound closed with deep catgut and horsehair skin sutures.

Here is what occurred in that original injury. The head of the radius was luxated out of its normal position, lengthened as bone growth continued until it struck against the anterior surface of the lower end of the humerus when the elbow was flexed. That is why she could not flex it. We have not disturbed the external lateral ligament nor the orbicular and oblique ligaments. The attachment of the biceps tendon to the bicipital tuberosity is also intact. We have shelled out the head of the bone. Here is what I take it has occurred. The head of the radius was thrown out of its relationship to the humerus and the ulna when she was five years of age. It irritated the side of the coronoid process of the ulna. It looks to me, although that is not clear, that there had been some elongation of that process.

She will be examined again at the end of four weeks. The wound will be healed so that we can make a good examination as to the degree of flexion and extension. If flexion is not so good as we think it ought to be, we will remove all or as much of the coronoid process as is necessary. The usual dressing will be applied and the arm carried in a sling.

[Stitches removed on eleventh day. Primary wound healing. Motion in elbow was limited—range of 40 degrees. Patient was instructed to continue active motion and to return in four weeks. When the patient returned, she had fairly good extension, but flexion was limited to about 80 degrees. The skiagram made at this time showed that the enlarged coronoid process was limiting flexion. A second operation was advised to be done in two weeks.—Ed.]

(Second operation June 26, 1913.)

Dr. Murphy: You will remember that we took out the head of the radius at the first operation, but we did not interfere with the coronoid process at that time. Why? Because if two freshened bony surfaces were in contact we might have an ankylosis of the head of the radius to the coronoid process. See the point? If I did both of these operations at one sitting,—and both were unquestionably indicated at the first operation,—I would have had a bony union. Now I am going to take out this elongated coronoid process because the skiagram shows that it is preventing full flexion.

We are told, in the discussion at the Minneapolis meeting of the A. M. A., that the results in fractures of the elbow had been satisfactory. If that were so, where would I get this colossal amount of material that is pouring in on me every day. I think that some cases of fractures in the elbow-joint are the hardest fractures in the entire body to control, because we are never just sure of them. That is why we worked out the extra-articular nailing scheme of handling these fractures of the elbow-joint by a purely mechanical fixation. A fracture of both condyles is common. In those cases reduce the fracture with the elbow in full flexion, and you get a good result. Sherman, of San Francisco, did not think so. Dr. Lee, my old partner, who had one of the largest emergency fracture practices in Chicago in his day, got results with that dressing. I got them in my early fracture work because then I got the primary cases—now I rarely ever get a primary case. Our plan, as we brought it out in the paper read in Minneapolis, is to control these fractures without opening the joint. It is not difficult at all. A considerable portion of both condyles is outside of the joint, so that you can treat these fractures as I do with the greatest ease, and without ever getting into the joint at any time. Make a small incision over the condyle, then drill a hole into it, directing the drill upward through the neck of the condyle toward the shaft of the bone, and drive in a small nail. That does not make a compound fracture; the only hole in the skin is where you have buried the head of the nail. It is much less difficult than to put on a Lane plate, and less dangerous. Nobody can put on a plate like Mr. Lane can. Not all of us can put on plates in an aseptic way, but we can all drill that bone and put in a nail if we are accustomed to doing aseptic surgery.

In the intercondyloid fractures you can also drill clear across both condyles and put in the nail without making a compound fracture or getting into the joint. All you have to do is to drill a hole straight through and put in a nail. I have been accused of being fond of nails. I am, because their use has always given me good results, better, in fact, than I could get by any other means of fixation.

You have to immobilize these cases absolutely. That was one of the things brought out in the discussion. One of the most important things is the necessity of absolute immobilization in fractures until the fragments have united, because every motion increases the amount of callus and of connective-tissue production. Let the limb remain absolutely still until union has taken place, then commence motion, but never to the degree of producing pain.

This woman has a limitation of motion now from two things: first, a limitation of motion caused by the pressing of the tip of the coronoid process against the anterior surface of the humerus; second, from the thickening of the posterior capsule. When a joint has been immobilized for a long period of time, the capsule shortens. The capsule does not limit motion, except when the motion exceeds the normal anatomic or an acquired pathologic range. If I flex my knee to the anatomic range of flexion, the capsule gives no support to the joint, but when I go beyond that range it does limit motion because it is inelastic. When you stretch it beyond a certain point, it will tear or fracture. In this case we will probably have to tear the capsule. It has been shortening for many years, and in order to get a full range of motion in the joint we must rupture it. Exposure of the coronoid process is not a pleasant task because we are in the range of the arteries and nerves near the elbow. You can reach it through an incision along the supracondyloid ridge and come onto the surface of the humerus. That is the way we do when we cut a contracted capsule where there is no elongation of the bone at all.

Here flexion and extension are limited. Note how you can hear the coronoid process strike the humerus. There is the ulnar nerve. It is back of where we are going to work. There is the supracondyloid ridge. It does not go straight up—it goes inward and up, therefore if we make our incision parallel with the long axis of the arm, we are likely to come onto the ulna. You cannot go through that great belly of muscles to the coronoid process. If you do you will injure the muscles, but you can go in from above, by going under the brachialis anticus.

We will not put on a constrictor this time, because we want to have all the bleeding stopped before we are through with the operation. If you put on a constrictor, the oozing continues after the operation and a hematoma may form. A hematoma is always an element of danger in bone and joint work.

The vascularity of the elbow is shown beautifully in Dr. Hochrein's skiagrams. (See August, 1913, CLINICS.) We have had the vessels injected with red lead and then made a skiagram of every large joint. That is far superior to a dissection, because it shows every twig of every vessel and shows you from where the tissues get their blood-supply.

We make a straight incision parallel to the anterior portion of the internal supracondyloid ridge. We retract the skin-edges and displace the brachialis anticus muscle inward. There is the coronoid process in the field.

When I flex the elbow now you can see that this process is striking against the humerus. We will remove that exostosis. I want to remove the periosteum with the bone, because otherwise we will have a callus formed. If you leave the periosteum attached to the bone at one end and the other end elevated, you will have a callus form beneath it. We will remove that part of the coronoid process with the narrow, half-inch, straight carpenter's chisel. There is little danger of injuring any vessels or nerves here. Now it is out.

There is the cavity left after its removal. Now, then, will the elbow come around? There is still the resistance of the capsule behind, which we must slowly free by forced flexion. That must be done slowly, and by interrupted flexions—not one continuous pull. I feel the capsule cracking. Now I have the forearm fully flexed. It comes straight without any trouble. That is all there needs to be done in this case.

Let the record show that we exposed the joint over the anterior portion of the internal supracondyloid ridge; that we then elevated the brachialis anticus and gradually progressed anterior to the capsule and periosteum covering the coronoid process; the bone clearly exposed to view; that the process was then removed with its periosteum at its base for the entire width with a straight chisel, and that then the elbow was gradually flexed to the full anatomic limitation. The arm will be dressed at an acute angle.

This wound is bleeding just a little more than usual, and for fear that a hematoma will form under the skin, we will ligate every bleeding point. Now we will close the wound. I unite the cut edges of the deep fascia with catgut so as not to put tension on the line of skin sutures. The skin wound is closed with horsehair.

You can see that in doing that work I never had any one of the important structures in the elbow in danger for one moment, because I followed a route which kept me in front of the joint until I saw the coronoid process.

[Stitches removed on ninth day. Primary wound healing. Range of motion better than after first operation. Patient left hospital and returned for examination after two weeks. She had almost full extension and free flexion to better right angle.—Ed.]

UNUNITED FRACTURE OF THE RADIUS, PREVIOUSLY PLATED; TRANSPLANTATION OF BONE

HISTORY

Patient, male, aged twenty-seven, comes to hospital on account of weakness in, pain and deformity of, the right forearm. June 17, 1912, the patient's forearm was caught in a machine. He had a great deal of pain in the arm at the time, with a swelling three inches above the wrist, and a false point of motion. A diagnosis of simple fracture of the radius was made. A splint was applied for five days. June 22, 1912, a Lane plate was put in, and the arm dressed without a cast. The splint was kept on for six weeks. The wound healed by primary union in ten days. He had much pain. The arm apparently was all right, except that it was not very strong. There was a slight deformity. Four weeks after the plate was put in he accidentally struck the arm on a screen-door and he thinks this pulled out a screw. The plate was removed the latter part of September (1912). The wound again healed promptly. Since that time the deformity has increased. The hand is deflected toward the radial side, and there is a large hard mass on the posterior surface of the forearm. He still has pain and slight motion at the site of the old fracture, three inches above the wrist. There is very little pronation or supination.

Past history negative; venereal infection denied.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 19, 1913): The skiagrams in this case tell a rather interesting story. They tell the story of the repair of bone very beautifully. One skiagram shows the Lane plate on the radius. There is a splendid apposition of the radius fragments. There is a perfect apposition of the ulnar fragments, and it seems that the patient had something like a "green-stick" fracture, with practically no displacement of the fragments. There is an effort on the part of the ulnar fragments to produce a callus. Periosteal callus is being formed. There is no effort on the part of the radius fragments to produce a callus. The fact that the Lane plate became loosened had little or no effect on the final result, nor did the second injury.

The third skiagram shows that he has a very good union of his ulna, but no union at all of his radius. There has been produced a large amount of bone around the ends of the radius fragments, but no effort at union. It is a typical pseudarthrosis. There is a subluxation of the ulna. The ulna is displaced to the right. There is an anteroposterior luxation of the fragments at the present time, a luxation backward of the upper end of the lower fragment, with a displacement posteriorly of the lower end of the upper fragment. That is going to be one of the difficult points in the management of this case, namely, to reduce the deformity and to get an end-to-end apposition of the fragments. Looking at the perfect apposition of the ulnar fragments in the early stage and at the practically perfect apposition of the radius fragments maintained by the plate, one naturally asks himself why it is that there was a non-union. Why did these fragments not unite? Not because there was a displacement of the fragments, because they were in good apposition. There was too much immobilization. We know that when the fragments are much displaced, even very much displaced, union will often take place. We see examples of that type of fracture every day where there has been very great displacement of the fragments, and still a bony union has occurred. It seems to me that this is one of the greatest evils that we have to encounter at the present time. If I were asked the question, "Do we have more ununited fractures today than we had a third of a century ago?" I would have to answer with an unqualified "Yes." It is not a creditable thing to say, but it is based on my observations in a ten-year attending surgeon's experience in the Alexian Brothers' Hospital

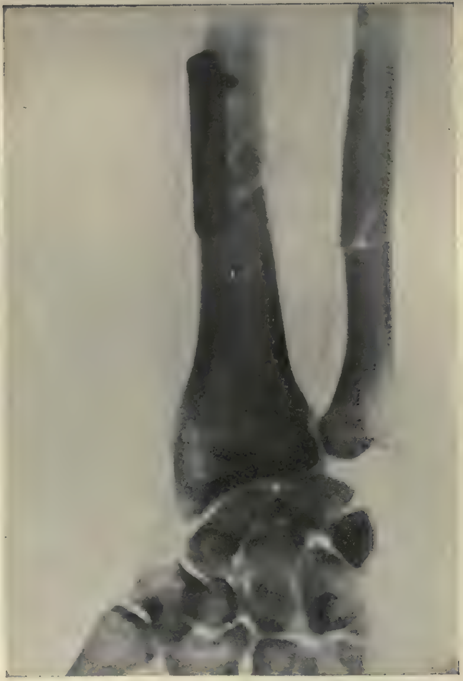


Fig. 243.—Ununited fracture of the radius. Anteroposterior view, just after first operation. Lane plate on radius; perfect alinement and good apposition of both bones. Only two screws were put in the plate.

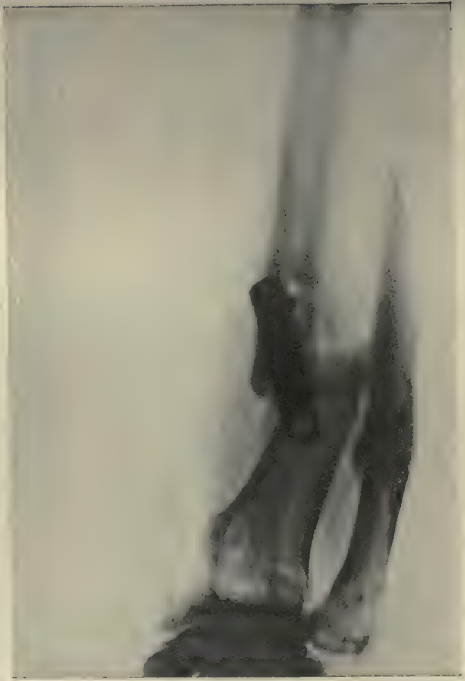


Fig. 244.—Ununited fracture of the radius. Anteroposterior view, made just before second operation. The ulnar fragments have united, with production of some excess callus. Bone fairly straight. Great displacement of radius fragments and excessive callus formation. The plate did not continue to maintain the apposition of the fragments because only one screw was put into each fragment and he had a second injury. The ulna does not articulate properly with the carpus.

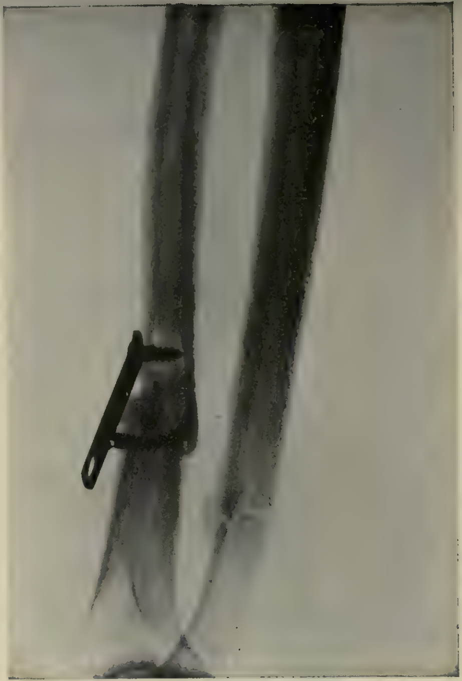


Fig. 245.—Ununited fracture of the radius. Lateral view made just before second operation, showing good alinement and apposition of the fragments. The Lane plate has been torn away from both radius fragments, especially the lower.

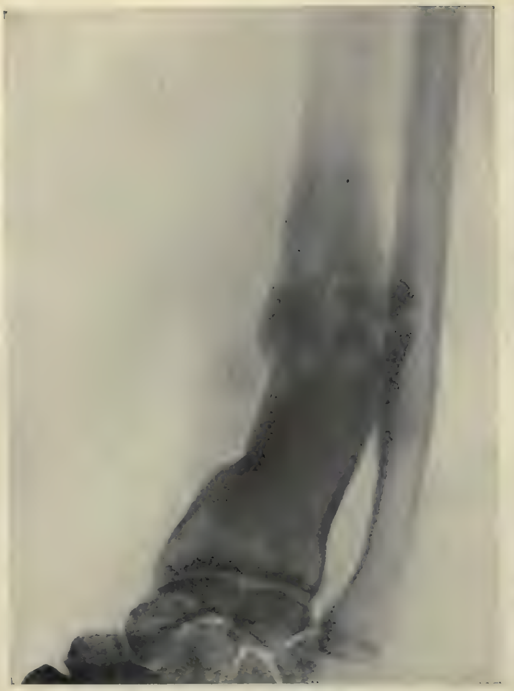


Fig. 246.—Ununited fracture of the radius. Anteroposterior view after second operation—removal of the Lane plate. The ulnar fragments are solidly united; those of the radius are ununited. Excessive callus.

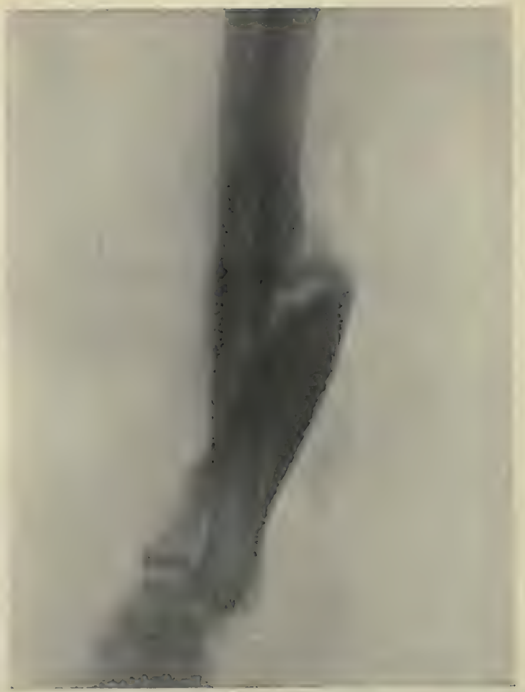


Fig. 247.—Ununited fracture of the radius. Lateral view after removal of Lane plate, showing the non-union of the radius fragments and tipping backward of upper end of lower fragment. The ulna does not articulate properly with the carpus. The end of the ulna is displaced backward.

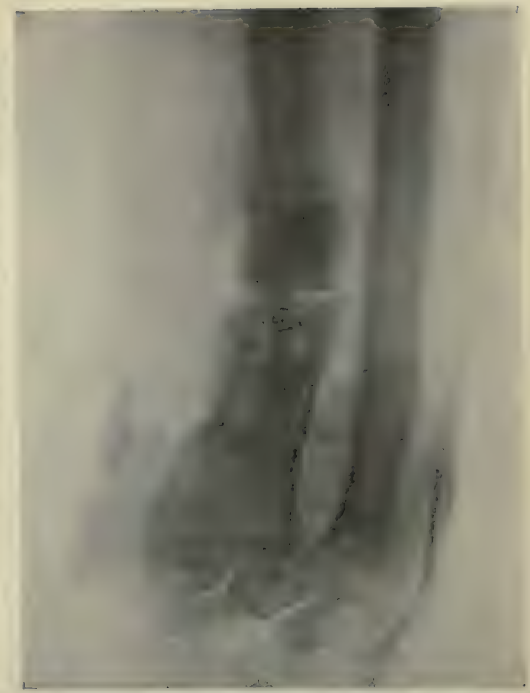


Fig. 248.—Ununited fracture of the radius. Skiagram made about four weeks after the transplantation. The alinement is nearly perfect. The transplant shows very distinctly in both fragments. The deposit of new bone may be noted on the ends of the fragments.

and twenty years in the County Hospital—the largest fracture services in Chicago.

The one factor that has contrived to produce non-unions more than anything else is the plaster cast. Plaster casts produce a too perfect immobilization of the ends of the fragments. Whether the interpretation is correct, I do not know, but the clinical fact is that the number of non-unions is constantly increasing. No matter whether we use a plaster cast or a Lane plate,—and Mr. Lane, in speaking of the use of the plate, said that it was not the best method of treatment of non-union—we must do something else to get a sufficient irritation at the point of fracture to stimulate the reproduction of bone. We know that in animals there is rarely ever a non-union after fracture. The conformation may be bad, but a bony union takes place. If that is true, then it seems to me that we will have to adopt the method used by the Germans for the past fifteen or eighteen years. When they put on a cast or immobilizing apparatus of any kind, they put the patient in ambulation at once. By the ambulatory treatment sufficient irritation of the ends is produced to favor bony union. We see many cases of perfect apposition of the fragments, with perfect alinement, and yet without the slightest effort at the formation of a callus. There was no osteogenesis, no effort at union, no effort at the production of bone, either by the periosteum or by the medulla or by the osteoblasts in the Haversian canals.

One of the easiest places to immobilize with a plaster cast is the lower third of the tibia. That is also the point where non-union occurs most frequently. It may be the condition of the vascular supply of the bone which produces it, although that is not a sufficient cause. In this class of cases the elements necessary to produce a union are lacking. If these fragments did not unite with as perfect an apposition as was shown to exist in the first skiagram, where the Lane plate is on,—and it remained aseptically fixed until the time that the arm was injured and the plate was loosened at the ends,—there was evidently something more needed than approximation and fixation. If you cannot get bone regeneration under such conditions, then you are not likely to get it under ordinary conditions.

The means we are using at present in the treatment of non-unions is to place a bone transplant across the line of separation, either in a groove or in the medulla, using a material that will stimulate the reproduction of bone. The best material we have found for doing this is bone taken from the same patient—an autotransplant. Bone taken from another patient—a homotransplant—lives if it is aseptic, but in a considerable percentage of the cases it is absorbed too soon and does not act as an osteogenetic conducting force. Bone taken from the ox, sheep, calf, or cat and transplanted into a human patient is always absorbed, even when contacting above and below with living bone. The only benefit one derives from such a transplant is that it is an irritant and immobilizes the fragments while it is in place. Bone taken from the same individual and put in aseptically and contacting with living osteogenetic bone, at one end at least, always lives. It is a little difficult to understand some of these statements, but if you will read carefully the observations of our experience and experiments, you will understand them.

We will make the incision in this case along the line of the old scar, so as not to disturb the tissues more than is necessary. The fracture is easily and quickly exposed.

Connective tissue is interposed between the fragments which caused the non-union. There was no connective-tissue interposition primarily, as was shown clearly by the skiagram showing the plate in position. This is not the same procedure which we employ in making a pseud-arthrosis. In making a pseud-arthrosis we endeavor to save all the periosteum on the ends of the fragments and use it as interposing tissue, capping the end of the fragment with it. In doing an arthroplasty we take it out completely, so as to prevent the growth of subperiosteal callus. Here the periosteum is of value, and we endeavor to leave it on the fragments to aid in producing a subperiosteal callus subsequently.

I am now freeing the upper end of the lower fragment and the lower end of the upper fragment where it contacts with the periosteum of the lower fragment. Here is the line of demarcation between the two bones. I will pass down on the inner side and displace the periosteum and endeavor to expose the lower end of the upper fragment. I am on the side of the bone. That brought me rapidly onto the upper fragment. I am leaving the periosteum. I am right on the radial artery. I am going to take off half an inch of bone from the end of the upper fragment, so as to give me a square surface. I am going to use a bone elevator as a lever and endeavor to see how much of the shortening I am going to be able to overcome. Dr. Edward Martin, of Philadelphia, taught us that when stretching a muscle we must go slowly—very slowly. It is really remarkable how much a muscle can be stretched in that way. In the majority of cases it works admirably. Now we have secured sufficient elongation. We will bring the tip of the lower fragment forward and take off as much of it as is necessary to secure apposition. The great difficulty in this work is to keep from contacting one's hands with the wound or with the instruments that finally touch the wound. The strictest asepsis is absolutely essential to success. The tendency is for these fragments to go forward; therefore I am going to put in an intramedullary bone splint. This is all compact bony tissue, the result of the inflammatory conditions that had previously existed. There is no medullary canal in that bone. We will chisel out a groove one inch long and half an inch in width from the end of the lower fragment, to facilitate placing the transplant.

We will take the transplant from the crest of the tibia in the usual manner. We do not divide the skin directly over the crest of the bone, but a little to the outer side, over the tibialis anticus. The aponeurosis of this muscle is split, and the muscle is retracted downward so as to expose the crest of the tibia. The periosteum is divided and the bone is nicked above and below to mark the the length of the transplant we wish to remove.

Formerly we used the chisel to take out the transplant, but we do this much more quickly and with less effort with the Zapffe buzz-saw attached to the Neff craniotomy motor. We will remove a piece about 2½ inches in length, ⅜ x ⅜, with its periosteum. The split muscle aponeurosis will be sutured with catgut and the skin with horsehair. This gap in the tibia will fill in very quickly with new bone.

We will now place the transplant. That little groove in the lower fragment helps us to insert the transplant into the medullary canal. We drive it into the medullary canal of the upper fragment for a distance of about 1½ inches, and then slide the lower end into the groove made in the lower fragment. We grasp the transplant with sequestrum forceps and drive it down into the medullary canal of the lower fragment. Both canals are first enlarged with our special reamer. It is a simple mechanical procedure, but it is difficult to describe in words. That practically completes the operation. The muscle aponeurosis is sutured with catgut and the skin with horsehair, just the same as was the procedure in the case of the leg from which the transplant was removed.

The important essential in the technic is to keep the hands from contacting with the wound surface or an instrument, except where it must be held. In this whole manipulation we did not contact a single instrument with the fracture which had previously been in contact with the hands. That is the secret of Mr. Lane's success in his bone work. Here I was dealing with compact bony tissue, and that is not nearly so easy to infect as cancellous tissue.

The rôle which that transplant plays in this particular case is a double one. First, it immobilizes the ends of the fragments and prevents them from sliding out of position as the bone grows. That could be accomplished with ivory or bone of animals, as well as with human bone. But these materials would play no rôle in the regeneration, while in this case you have put in a mechanical immobilizer of bone taken from the same patient. The rôle it plays is this, as far as we are able to figure it out from the microscopic reports and the skiagrams, which we believe add much light to the process of regeneration: the Haversian canals in this transplant permit the vascularization from the neighboring connective tissue, where the ends are both contacting with the medulla of the living osteogenetic bone. In the study of the regeneration of bone, if you just liken the osteogenetic areas to a tree, you will get an idea of all the elements that enter into the regeneration of bone.

The medulla is the trunk of the tree. In it are situated the osteogenetic elements—the osteoblasts. They extend or travel into the trunk, limbs, and branches. The blood-vessels from the living bone make their way into the Haversian canals of the transplant. On the walls of these vessels are osteoblasts. They also go into the Haversian canals of the transplant. The osteoblasts and the blood-vessels carrying them are the osteogenetic forces. The transplant is osteoconductive. The periosteum, in its lower layers, carries along with it osteoblasts, and these stretch out on the surface, resembling the roots of a tree, small in number in the adult, larger in number in the young. Whether you say that this layer is the outer layer of the bone or that it is the inner layer of periosteum, is purely a matter of nomenclature. Technically, osteoblasts are situated there. Whether it is true, as Macewen believes, that there are no osteoblasts in the periosteum (he says the osteoblastic layer is beneath the periosteum), or whether they are, as others believe, a part of the periosteum, does not matter to the surgeon at all. What we want to know as surgeons is the practical side, namely, that the regeneration takes place; that the osteoblasts are present; that a callus is formed, and that new bone is formed. We want to know what we can do to stimulate or to favor osteogenesis.

Is it necessary that one should have a transplant with the periosteum attached to it? I think not. Do I leave it on the transplant? Yes, because I am not convinced that it does not play a rôle. Why should I hazard my result by experimenting? We have often watched the periosteum being stripped off the transplant as we were driving the latter into the medullary canal of the fragment. Practically only that portion of the transplant which is exposed and free between the fragments, that is, in the gap between them, is still covered by periosteum, and only shreds at that. It matters nothing to the patient or to me if I leave the periosteum on the transplant. In some of our cases it appeared to be a detriment to the process of repair that it did not permit of the unrestricted regeneration of bone. We do not believe that the bone-cells, the lacunæ, and canaliculi play any other rôle than a mechanical one. That is the proposition. The osteoblasts alone are concerned in osteogenesis.

This arm will be dressed at right angles and placed in a cast which will be split immediately with the Gigli saw, *with perfect immobilization of the hand, forearm, arm, and elbow. When a bone transplant is used in non-union, absolute immobilization is essential to the development or passage of the Haversian vessels from the living bone into the Haversian canals of the dead transplant. If motion is permitted, a fibrous tissue develops between the transplant and the living bone and a pseudo-arthrosis may result, or bony union may be retarded. The process here is entirely different from that in the union of simple fractures, where limited motion is desirable, as it favors ossification.*

[Stitches removed on twelfth day. Primary healing. Patient left hospital at end of fourth week, with arm still in a cast. He returned for examination after three weeks with an apparently perfect arm.—Ed.]

ANKYLOSIS OF ELBOW

HISTORY

Patient, male, aged twenty, comes to hospital on account of inability to move left elbow-joint. Trouble dates back to September, 1912 (first five days of month). One cool evening patient went out without overcoat and became chilled through and through. He shook for from five to ten minutes with the cold. The next morning his elbow was stiff, and when his room-mate rubbed it, it became painful. That afternoon the joint became swollen, red, and tender to touch. He could not move it on account of the pain. He says he felt hot, but his temperature was not recorded. During the first few days he had a temperature of 101° F. Two and one-half weeks after the onset of the trouble the arm was put in a plaster cast at greater than a right angle. The cast was taken off at intervals during the next two months. Patient states that there was motion in the elbow at these times. Three months later when the cast was finally left off, the elbow was stiff, but the swelling had disappeared. At present he has no pain or tenderness. Did not have sore throat or cold preceding onset of trouble. Four days before onset he began to have a urethral discharge (incubation seven days). After a week the discharge stopped for four or five days, then appeared again, and continued for six weeks. He has been having a urethral discharge now for four days. Otherwise venereal history is negative. Had pneumonia four years ago; scarlet fever nine years ago. Bowels constipated; no night-sweats or afternoon rise of temperature.

COMMENTS AND OPERATION

Dr. Murphy (Operation April 9, 1913): What year did the trouble begin?

Intern: September, 1912.

DR. MURPHY: Has the pain disappeared?

INTERN: Yes, doctor.

DR. MURPHY: That means what?

INTERN: A bony ankylosis.

DR. MURPHY: Yes, otherwise he would still have pain. Remember that when the cast was finally taken off the elbow was immobilized and painless. The "painless" tells the story better than anything else. It may be immobilized by the voluntary contraction of the muscles, but when he tries to flex the arm, the motion is gone. Did he cough or expectorate at this time?

INTERN: No.

DR. MURPHY: Was the sputum examined for pneumococci?

INTERN: No.

DR. MURPHY: In every case where there is a history of a pneumonia preceding the arthritis the sputum should be examined to see whether pneumococci are still present. We had a case of that kind—rather a painful experience—in which we operated on a doctor's son. He got a hematogenous pneumococcus infection after an arthroplasty on the hip. He had pneumonia when he had his original infection in the hip. The pneumococci were present in the sputum, as they are permanently in 60 per cent. of the patients who have had pneumonia. Fortunately, the final result was not hazarded in that case.

This boy "caught cold" in his elbow. That is what the preceding generation would say. That is all right. That explains the condition. What started the pain in the elbow? He had an active Neisserian infection at the time of the exposure, and he had a metastatic deposit in his elbow, correlated with the exposure. I believe the exposure is the etiologic factor, but only of secondary importance. This case is exceptional, and I put a very large, black interrogation point after his statement that he had his Neisserian infection only five days before. Adding to it the period of incubation, which he said was seven days, making twelve days, it is still too close to the joint infection. I think that his estimates are wrong, because in our cases, which now represent a very large number, the metastatic infections

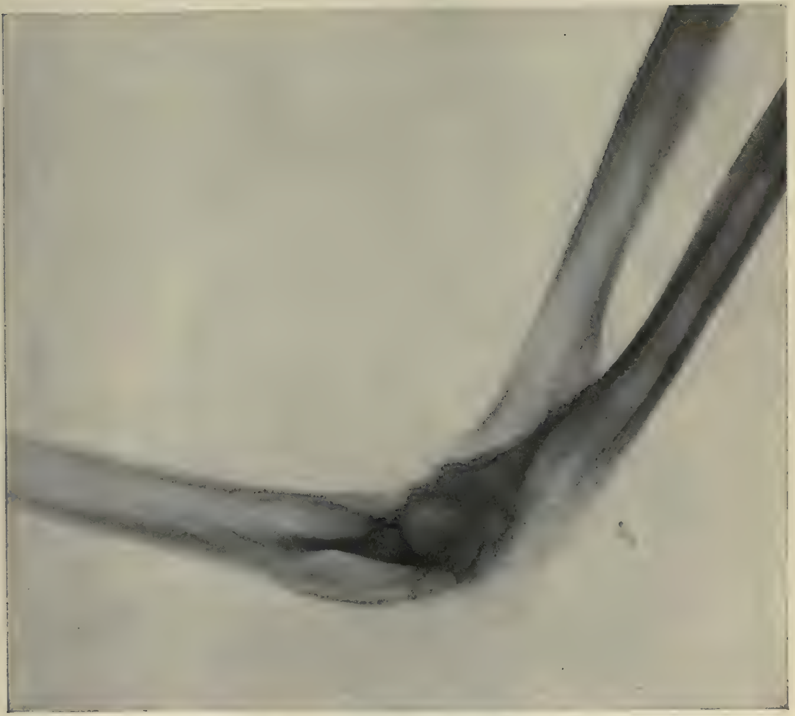


Fig. 249.—Ankylosis of the elbow. Lateral view made before operation, showing a complete obliteration of the joint-cavity by a bony ankylosis between the humerus and the ulna.

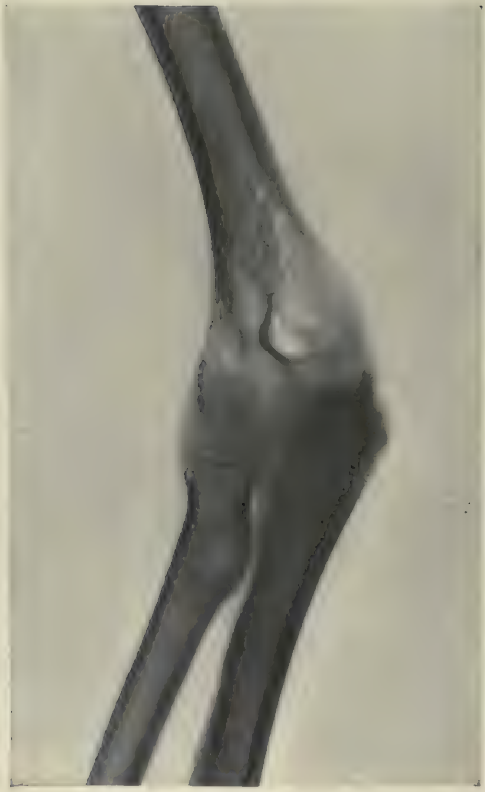


Fig. 250.—Anteroposterior view, showing complete union of the humerus and ulna, with the dim outline of the cavity between the radius and the humerus still apparent. There is a considerable new formation of bone around the upper extremity of the ulna.

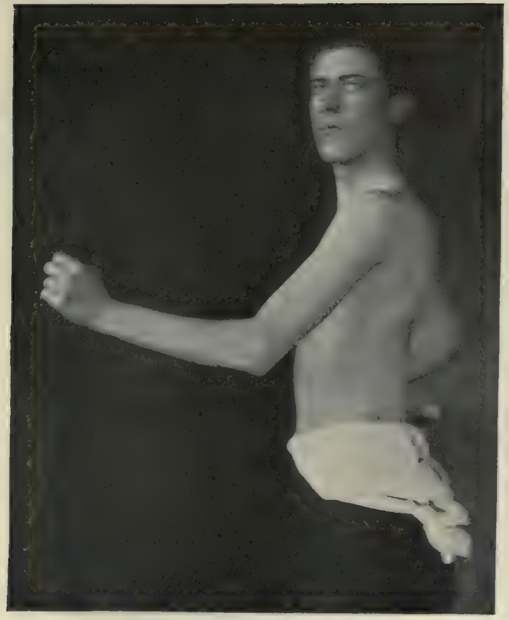


Fig. 251.—Photograph of patient made before operation, showing the arm flexed at the elbow at an angle of about 100 degrees.

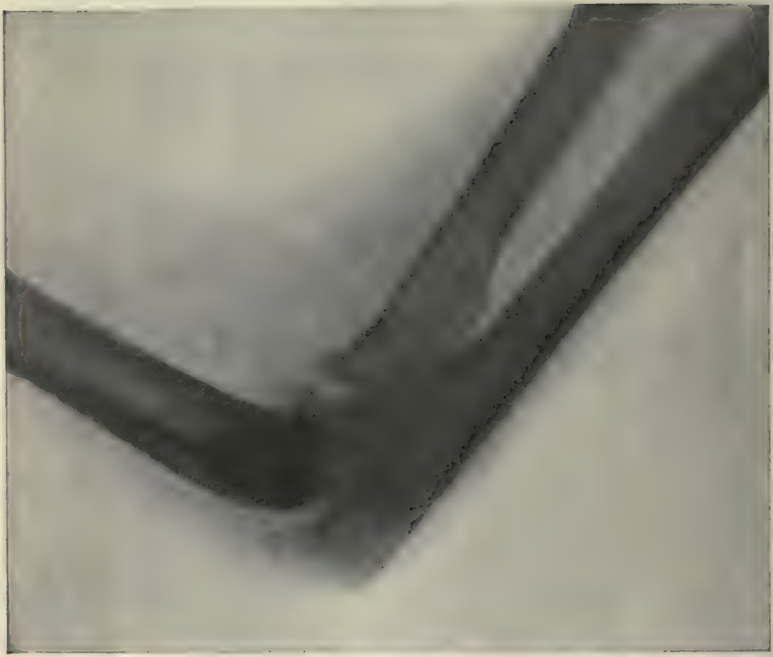


Fig. 252.—Lateral view of elbow made about two months after operation, showing a nearly normal joint-cavity. The upper end of the ulna has been reformed. The radius occupies its normal position. The patient had fair voluntary motion in the joint at this time.

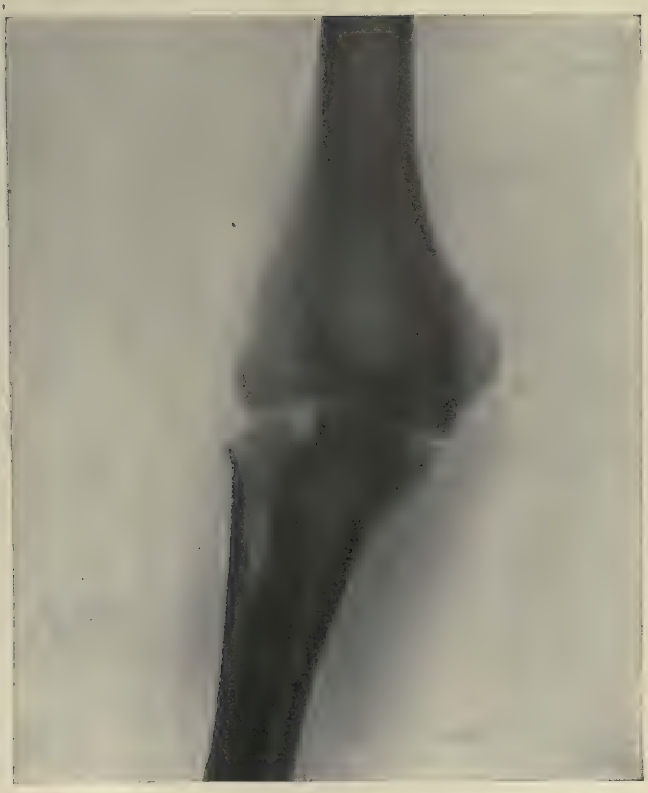


Fig. 253.—Anteroposterior view, taken about two months after operation, showing restoration of bone cavity. The radius articulates normally with the humerus.

from the urethra take place in an enormous percentage of the cases—we have estimated it as 96 per cent.—between the eighteenth and the twenty-second day. The fact that the joint metastases take place so uniformly as to time shows what a prominent part of the regular pathologic process that is, and that the exposure is merely a coincidence, because the exposures that occur on the fifth or sixth day or on the thirty-sixth or fortieth day do not give these metastatic infections. In reality, the great majority of gonorrheal joint metastases occur between the eighteenth and the twenty-second day after the appearance of the discharge.

This case went the typical course of a severe metastatic infection. How could we have known the very first day of the arthritis that that elbow was going to become ankylosed? First, by the fact that the infection involved only one joint, and, second, that its initiation was accompanied by a chill. Whenever the arthritides are preceded by a chill, no matter whether it is one or many or a small number of joints, the rule is that an ankylosis will occur if proper treatment is not instituted at once. In the arthritides that are not preceded by a chill, showing a lesser degree of virulence, the ankylosis is not so apt to occur. No matter what the treatment is, the clinical fact remains that an arthritis not preceded by a chill rarely ever results in an ankylosis. Our forefathers were right about that. I remember when I was an intern, thirty-five years ago, hearing Dr. J. H. Hollister talk about the severity of rheumatism. He divided it into two classes—those cases that were preceded by a chill and those that were not. He said that the class that did not have a chill preceding was not pyemic, and therefore not so trying on the joints as was the class that did have the initial chill. We know now he meant that they did not lead to a destruction of the joint and recovered more rapidly.

Remember, every case of acute arthritis is a metastatic infection. We believe that in the large percentage of the cases you can determine the exact primary focus and the atrium through which that infection was admitted into the circulation. The result of the infection in this case was a severe inflammation in the joint, and, with the assistance of the doctor, who put on a perfect immobilizing splint, the ankylosis resulted. In acute inflammatory conditions in joints, if you will separate the joint surfaces by putting an extension on the limb, you will lessen the likelihood of an ankylosis occurring, while if you put on a cast, you will favor ankylosis. On the other hand, if you absolutely immobilize a tuberculous joint, you lessen the likelihood of an ankylosis occurring. Because the orthopedists used plaster casts in tuberculosis and avoided ankylosis, the general surgeons began using them for all forms of arthritis. The orthopedists had as their reward movable joints. The general surgeon had as his reward in the acute infections a fixed joint. Those are clinical facts.

The difficulty in doing this bone and joint work lies in the danger of infection from the skin. It is practically impossible to sterilize the skin. I have for many years advocated the use of a gutta-percha solution to cover the skin in the field of operation and believe it especially advantageous in joint work. You cannot put on towels and adequately protect joints from skin infection when doing this extensive work. A constrictor has been placed on the arm.

We will expose this joint by means of two incisions on the back of the arm—one on either side of the olecranon process, about 1½ inches apart.

This boy has very little adipose tissue, which is unfortunate. We like fat patients in these cases, because they gives us a better interposing flap. I will make two flaps,—an inner and an outer,—taking the deep fascia and some of the aponeurosis of the supinator longus muscle for the outer flap, and fascia and fat for the inner flap. The base of each flap is directed downward. These flaps are made wide enough to cover the freshened surfaces of bone, and long enough to reach from one side of the joint to the other. There is a complete bony ankylosis of this joint; it is perfectly fixed. We will have to take out a portion of the head of the radius. In the original operation we divided the olecranon. We do not do this any more; it is not necessary, except in unusual cases.

The next step is the exposure of the ulnar nerve. We always locate this nerve because there is danger of injuring it during the operation. We free it and displace it entirely out of our way. We do not put much tension on it, because if we do, we will have a temporary suspension of transmission and paralysis of the muscles supplied by it. There is practically no line of demarcation discernible between the bones of this joint. I am going to free the radius first, using a narrow, slightly curved chisel. Then I will free the ulna. Now you see the advantage of having the ulnar nerve out of harm's way. Now we have the ulna freed, by driving the chisel in between the ulna and humerus very carefully. Remember that this new-formed bone is cancellous bone and more easily divided than compact bone. We must take off a considerable portion of the lower end of that humerus to make room for the interposing flaps. We always endeavor to restore the normal conformation of the joint. The head of the radius is going to impinge against the humerus unless I take it off. We will also take off a portion of the external condyle of the humerus, deepen the olecranon fossa, and remove a small portion of the tip of the olecranon, as well as of the coronoid process. Ample bone should be removed to permit of free flexion and extension without force. I must take off a little more of the radius on the ulnar side, otherwise we will get a reankylosis between the ulna and the radius. You may think that we are going to have a flail-joint here. Not at all. We are going to have a sliding joint. Now I am preparing the interposing muscle and fascia flap to place between the head of the radius and the lesser sigmoid cavity. This does not in any way interfere with the muscle action. I just read a comment in a recent article in which the author thinks that this operation interferes with the musculature. It does not. We tuck that muscle and fascia flap down between the radius and ulna and hold it with one chromicized catgut suture.

Next we put in the interposing flaps, which will cover the entire articular surface of the humerus so the ankylosis cannot recur. These flaps are drawn into the joint and secured on all sides with interrupted chromicized catgut sutures. In that way all raw or freshened bony surfaces are covered over with a fascia and fat pad, and the ankylosis cannot recur. The ulnar nerve is now replaced in its groove and surrounded by fat.

Let the record show that there was a bony ankylosis between the olecranon and the humerus and between the ulna and radius.

We took off the constrictor early, so that all the oozing would stop, otherwise we would get a hematoma. We have found that a hematoma interferes with the smooth recovery of these cases. It prevents primary union, and early passive motion must, therefore, be delayed. A few deep catgut sutures will be put in to take the tension off the skin.

Let the record show that the line of union could not be discerned, except as an anatomic estimate; that there was nothing left of the joint itself.

The oozing has all ceased, and we have not put on a ligature. We will close these wounds with horsehair and dress the arm in right-angle flexion with the usual bismuth subiodid powder, collodion strip and alcohol, and phenol gauze. The arm will be carried in a sling.

The circulation in these periarticular flaps around the elbow is very good, and we rarely ever have a necrosis of the flap here. When we were making the "U"-shaped flap in the knee arthroplasty, we had a number of cases of necrosis of the tip of the flap and it retarded all efforts at motion. We deem it a little inconvenient, and I think it detracts from the results because we cannot institute motion as early as we desire. In this case we want to commence gentle passive motion at the end of two weeks, then gradually increasing it until we can put the joint through a nearly normal range of motion. We do not put any extension on in these elbow cases.

[Stitches removed on the sixteenth day. Primary healing. Slight voluntary motion in joint. This patient did not have grit enough to allow sufficient forcing of the joint by the masseuse or in the machine, so that progress was slow. However, two months after the operation he had a voluntary range of motion of about 80 degrees, and at this time it was increasing less slowly than it had been.—Ed.]

LAMINECTOMY FOR TUBERCULOMA OF SPINAL COLUMN, WITH COMPRESSION OF SPINAL CORD; KYPHOSIS AND LATERAL CURVATURE; MOTOR PARALYSIS

NEUROLOGIC FINDINGS BY DR. CHAS. L. MIX

HISTORY

PATIENT, a male, aged sixteen, enters the hospital because of stiffness of both ankles and knees and inability to walk. There is no pain or swelling. The onset of this condition was in July, 1912. It began with stiffness of the toes of the right foot, worse after standing or walking. He paid no attention to this at the time. In October, 1912, he noticed that the toes would jerk at times. About April, 1913, the stiffness started to spread upward, finally involving the entire limb. When walking, he had to drag that limb on account of the stiffness. About the latter part of June, 1913, the left limb became involved. He then was unable to walk. He was put to bed, and has been there ever since. During the past month and a half his legs and anterior abdominal muscles became very rigid at times, the rigidity lasting from a few seconds to several minutes, coming on about once every hour or two, and at times more frequently, especially when attempting to move the limbs. When this comes on, he abducts the limbs and the rigidity disappears.

At the present time he cannot raise either leg nor can he raise his shoulders off the bed without the aid of his hands. He never had any elevation of temperature nor chills and sweats at any time so far as he knows. A history of trauma is not obtainable; at least he cannot recollect ever having been injured. He had a slight cough, lasting several weeks, during the spring of 1913. He has had no pain or paresthesia at any time. He claims that he was perfectly well up to the onset of the present trouble, although his family told him two years ago that he had a deformity of the spinal column. He has no recollection of how or when this began. He claims that lately it has been hard to start his urinary flow. His bowels are constipated.

Family History.—Father died two years ago; cause of death unknown. Mother is living and well. One sister died of tuberculosis. Four brothers are living and well.

FINDINGS BY DR. CHARLES LOUIS MIX

Reflexes.—Patellar reflexes are markedly increased. The ankle-clonus was not obtained; however, the Babinski toe-sign is present on both sides. The epigastric and abdominal reflexes are absent. The plantar reflexes are present and exaggerated. The right cremasteric reflex is good; the left, much reduced, but present.

Paresis.—Spastic paresis of both lower extremities, almost to the degree of a spastic paralysis. The legs are extended and strongly adducted. The right big toe is cocked up in the position usually found in Friedreich's hereditary ataxia.

Bladder.—Control is present.

Rectum.—Enemas invariably required.

Atrophy.—None. Anterior horn cells not involved.

Sensation.—Reduction of the sensation of touch below the fourth right rib in front, extending around and upward to the level of about the sixth dorsal vertebra posteriorly. The level on the left side is lower by an inch than the level on the right side.

Diagnosis.—A compression of the cord, presumably a little more on the right side than on the left, but very evenly distributed, at the level of the fourth and fifth dorsal vertebræ. The cord is merely severely compressed, but not divided.

Comments on the Diagnosis.—The history of the case is extremely suggestive of a gradual compression from an infectious granuloma, in this instance caused by tuberculosis of the spine. It will be noted that the onset was in July, 1912, beginning with a stiffness in the right foot, worse on standing or walking. This is the first element of diagnostic value as to the localization in the patient's case. It marks the beginning of the compression of the pyramidal tracts. By October, 1912, just three months after the onset of the disease, involuntary spasms, the second step in the compression, began to appear. We note next that by the following April the stiffness had involved the entire limb, so that by this time, as the patient states in his history, "when walking he found he had to drag the limb." He said he had to drag the limb "on account of the stiffness." This was the way the patient expressed the fact that he was suffering from a spastic paresis of his right leg. By June, 1913, eleven months after the onset, the *left* limb was entirely helpless, so that the patient was paralyzed. Since August 1, 1913, the history states that there have been spasms involving not only the legs, but also the muscles of the anterior abdominal wall, thus clearly indicating a level as high as the innervation of these muscles.

The key to the localization of the process is furnished by the sensory symptoms. The upper level on the right side, which is the side of the greatest compression of the cord, is as high as the fourth rib. The area of sensory disturbance on the left side is at the level of the fifth rib. The sensory changes which are present are slight. They consist not in the entire loss of the tactile sense; for example, the patient feels, but immediately confesses that he does not feel touch as calling forth "natural" sensation below the level of the fourth rib. Above the level of the fourth rib he records all touches of the fingers as perfectly "natural."

In looking over the history, which extends from July, 1912, to September 20, 1913, it is evident that there has been a very slowly increasing compression of the spinal cord at the level of the fourth and fifth dorsal vertebræ, as high as the fourth on the right side and about on the level of the fifth on the left, making an apparently oblique compression of the cord. There being no atrophy of the muscles of either the right or left leg, it is evident that there has been no severing of the anterior roots and no destruction of the anterior horn-cells. The presence of the reflexes and spasticity is good evidence that the cord is merely compressed and not divided or degenerated. Such a slowly increasing compression of the cord is brought about, as a rule, by a tumor mass of some sort, the most common perhaps being an infectious granuloma. In this particular case a tuberculosis of the bodies of the fourth and fifth dorsal vertebræ has been responsible for the infectious granuloma pressing the cord backward against the laminæ.

COMMENTS AND OPERATION

Dr. Murphy (Operation September 20, 1913): Basing an opinion on that history alone, not having seen the boy, what would you think was the matter with him? I cannot form a clear conception of what is the matter with him, and I wondered whether you gentlemen came to any different conclusion from mine. That he has an interference with the transmission of motor impulses in his cord in the upper dorsal region is clear enough, but what is causing that interference? What would you say that interference was due to, judging from that history alone?

Visiting Doctor: He might have had an injury of some kind—a fracture, or a necrosis, or something of that kind.

Dr. Murphy: That is just what I want to bring out. Was there not a trauma at some time? That history does not tell the story at all. It does not tell the story of a beginning deformity in the region of the fourth, fifth, and sixth dorsal vertebræ, nor of the progress of that deformity to a superlative degree when he showed evidence of a motor paralysis in the leg muscles. You will note that, as a tail to that story, he added the fact that his folks told him two years ago that he had a deformity of his spine. That was the beginning of the trouble, and that should have been the beginning, and not the end, of the clinical history. [The patient is brought into the clinic.] Now that the boy is here, let us look at him. You will note that he has a posterolateral curvature of the spine in the upper dorsal region. The skiagram shows that the fourth, fifth, and sixth dorsal vertebræ are involved, with absorption of the anterior and left lateral portions of the bodies of the vertebra. There is a pronounced gibbus opposite the fourth dorsal vertebra. As a sequence of that he

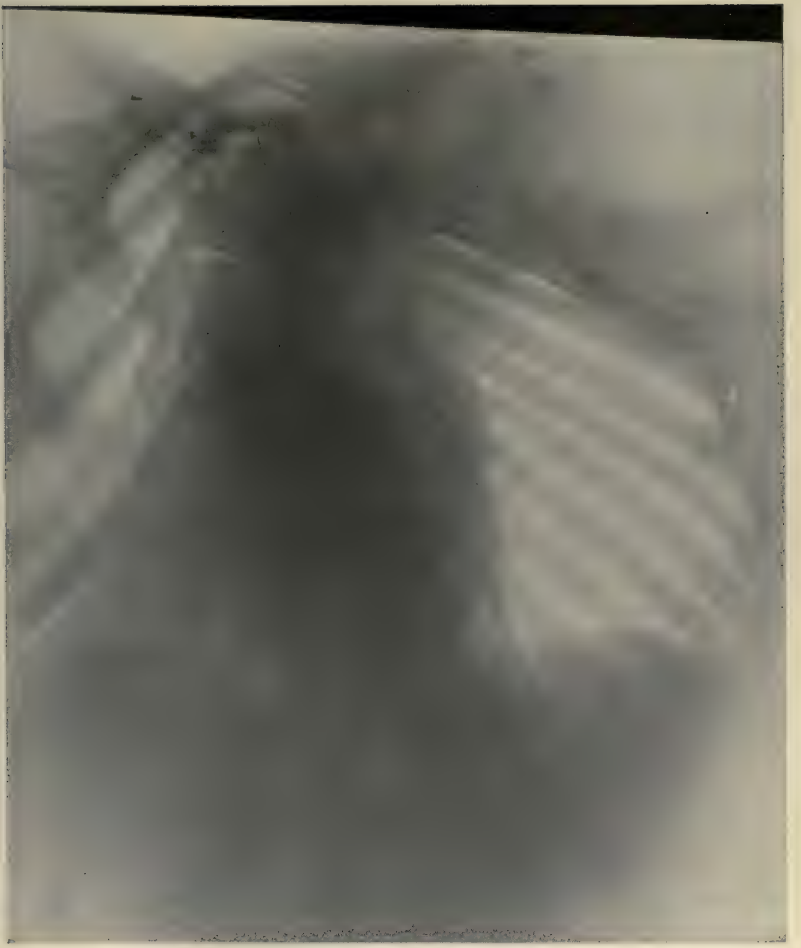


Fig. 254.—Tuberculosis of bodies of fourth, fifth, and sixth dorsal vertebræ, with kyphosis and left lateral angulation. The disease has invaded the anterior as well as the right lateral portion of the bodies.

has this motor paralysis of his legs, caused by the compression of the cord in front. Now the question is, what is the matter with him? He gives no history of trauma. "No history of trauma" does not mean anything. The intern did not make the proper kind of an investigation as to whether the boy had a trauma. If he had, he would have learned that the boy did have a trauma some time before the onset of this trouble. I have not gone into this phase of the case, but I am sure he had a trauma. Whether he fell and struck his shoulder or landed on his buttocks, or whether he was thrown backward or forward, or whether his body was bent laterally, must be finally determined. I would say that it was through some sudden, severe hyperextension of his body or that he fell on one shoulder and bent his spine sideways, and that the injury was a lateral injury to the body of the vertebra. Why do I say that? Because the primary curve in this case is a lateral curve, and the concavity of the curve is always on the side of the vertebral body that is traumatized. It is a tuberculosis of the spine, a Pott's disease, which always begins in the body of the vertebra. It is not an osteomyelitis, because the disease came on so slowly; that slow course is typical of a tuberculosis of the body of the vertebra. Pott's disease always begins in the bodies of the vertebræ. The laminæ and spinous processes are never primarily involved. We know that the disease begins on the side on which the concavity finally appears, because the absorption of the bodies of the vertebræ on that side is what causes the lateral bending. The disease begins more commonly in the anterior than in the posterior portion of the body of the vertebra, and in the posterior more commonly than in either of the lateral portions, because superlative traumas are more commonly anterior and posterior. We much more frequently hyper-bend than hyper-extend our bodies by trauma. Trauma is always an important factor in youngsters of this age, who are subject to the presence of tuberculous microorganisms in the lymph-nodes, because, as a result of such a trauma to the spine, the bacilli are metastasized or localize in the bodies of the vertebræ and then follows the destruction of this cancellous tissue.

Let me cite a case to give you a clean-cut idea how important it is to interrogate the patient carefully as to the occurrence of a trauma. A man came to me from Ireland several years ago for a diagnosis of a trouble he had in his back. He had had pain in his back for two years, and his back was fixed at the junction of the lumbar with the dorsal spine zone. I asked him whether he had ever injured his spine. He was positive that he had not. He had a lordosis—meaning what? That the disease involved the posterior portion of the body of the vertebræ. Remember that the concavity is on the side of the body involved primarily. Finally I elicited this story: One night, several years before I saw him, he went out into his stable without a light. The children had tied a rope across the stable door, and he fell over it, landing on the floor face down, so that he was bent double backward. He got a severe pain in his back. What portion of the vertebral body would be injured when he was doubled backward? The posterior portion. He had no paralysis at that time. The pain quickly subsided, and as he had no further symptoms, he forgot all about the incident. We made a tuberculin test and got a positive reaction. We put him to bed at rest. He went on and developed a lumbar abscess, finally developed a spinal curvature, and went through the typical course of a mixed infection with a tuberculosis involving the posterior portion of the body of the vertebra. I merely cite that as an illustration of the fact that trauma is the important etiologic factor in these cases.

This boy undoubtedly had his spine bent to the left side, because the softening of the bone came on the side on which he had the greatest traumatic pressure due to the lateral bending. Let us follow the case along. The intern began at the wrong end. He began with the paralysis. That would lead one to think that this is a spinal cord lesion—a spinal cord tumor or something in the spinal canal pressing on the cord. It is not a spinal cord lesion, it is a spinal column lesion. In a case with a curve of the spinal column as marked as this one is there must be something in that history to tell a man in Africa or China who would read it how the curve began—what caused it. That history should give some insight into the sequence of the pathologic conditions present, but it does not. What happens in these cases? What produces the paralysis? The paralysis is not due to pressure from the acuteness of the curve when it is an anterior or a posterior curve. It is sometimes due to a sliding of the bodies of the vertebræ, but that is rare. It is never due to a kinking of the spinal column diminishing the lumen of the tract. In the enormous majority of the cases it is due to the formation of a granuloma, that is, the tubercular débris penetrates the posterior wall of the body of the vertebra and encroaches on the lumen of the spinal canal as a tumor. That is what compresses the cord. It carries in front of it the fibrous tissue that lines the anterior portion of the spinal canal. That fibrous tissue is not often penetrated by it, because, remember, fibrous tissue resists the invasion of the tuberculous granulation tissue very materially, so that as the tuberculoma increases in size it compresses and flattens the cord. The bulging into the spinal canal of this tuberculoma or brie accumulation is what produces the paralysis. It slowly compresses the axons and cells of the cord. Dr. Mix, in his report of the findings in this case, says that the anterior cells of the cord are not disturbed; that is, they are not degenerated or dead; therefore the disturbance present is one of transmission, either through a degeneration of the axons that come down from the upper motor zone or a disturbance of transmission of the tracts that come from the anterior roots of the cord below the point of compression. Is it a complete degeneration? No. If it were a complete degeneration, then we would not be following out the plan we intend to follow this morning, because once the axons of the spinal cord are sufficiently compressed to cause a degeneration, then it is useless to operate, because the axons in the spinal cord, once they are degenerated or divided, are never capable of regeneration. That is a law.

The compression of the cord in this case may be due to one of two things. There is apparently a lateral sliding of the body of the fifth dorsal vertebra, so that there must be some lateral compression of the cord from the bones. If the bone is compressing the cord laterally, I will take out the spinous processes and the laminæ of the involved vertebræ and expose the cord. If there is a granuloma compressing the cord from in front, what will I do? Formerly we used to expose and clean out the granuloma by curetage, inject iodin or iodoform emulsion, and close the wound. A large percentage of the cases did very well, but a smaller percentage of the patients got an acute tuberculous infection in the tract, which invaded the neighboring tissues, penetrated the membranes of the cord, and finally caused a permanent paralysis of both sensation and motion, extensive bed-sores, and death. Therefore that is a procedure that we have no right to use.

Our plan in the management of this class of cases now is and has been for some time past to relieve the cord of the pressure on it by taking out the spinous processes and laminæ of the vertebræ involved in the compression and permitting the cord to be displaced backward, that is, doing a spinal decompression, and continuing the treatment of the patient with tuberculin so as to heal the tuberculosis. That is the safest plan. That is what I would want done if the disease were in my body, because I believe it is the safest course for the patient.

This is a very unpleasant case for operation on account of the lateral angulation. The operation which we perform in this class of cases consists in the removal of the laminæ and spinous processes so as to free the cord of pressure. It is a typical spinal decompression. We do not restore these bony structures, and it does not seem to have any ill effect on the final stability of the spine. [The patient is brought in ready for operation.] You see the patient is lying on his left side with the right knee drawn up as high as possible and flexed. The left leg is straight. That places the back in the most desirable position for operation, and respiration is not hindered or made difficult because the chest is free. The arms are in front of the body—not folded under it. The skin has been painted with half strength (3.5 per cent.) tincture of iodin, and the sheets and towels are in place so that only the field of operation is exposed.

We make a longitudinal incision directly over the spinous processes, extending from the third to the seventh dorsal vertebræ.

The soft tissues on both sides of the processes are retracted, and with the periosteotome the spinous processes of the fourth, fifth, and sixth dorsal vertebræ are stripped clean. The lateral angulation is now very apparent. No effort is made to preserve the periosteum in exposing the spinous processes.

With the rectangular bone-cutting forceps the spinous processes of the fourth, fifth, and sixth dorsal vertebræ are removed close up to the laminæ. That is done very quickly. Next, I bite out the laminæ of these vertebræ with the small rongeur forceps. That is very tedious work and is accompanied by considerable bleeding from the bone. These veins bleed profusely, but by a little compression and patience the hemorrhage finally stops. I will wait until that bleeding stops before proceeding. That bleeding is from the bone. We make pressure on the bone with gauze sponges. I am not pressing on the cord. I am pressing laterally, not downward. I will stop the bleeding with a little wax. That is one of the valuable aids that Horsley gave us. Small pellets of sterile wax, softened in hot water for a few moments, are pressed against the bleeding bony surfaces to check the hemorrhage. We always use it in these cases, and with uniform success. The bleeding being checked, we continue with the operation of freeing the cord from pressure. Grasping the body of the fourth dorsal vertebra I find that it is quite movable laterally. The cord is now partly visible. It is full and rounded above the fourth dorsal vertebra and pulsates, but there is no pulsation of the cord below this vertebra. That is the point where the cord has been compressed, and there the cord is also flattened out considerably. As soon as we relieve the cord of all the compression it will have room to expand backward. On account of the lateral angulation we are obliged to remove a great deal of bone. Now, however, the cord is free. We have not opened the dura at all, so that the cerebrospinal tract is in no danger of infection. Nor have we traumatized the cord. There is no need to go higher up, because I am above the point of compression now. There is sufficient room for the cord to expand backward. Even with so much bone removed the spinal column is sufficiently supported without resorting to artificial means of support.

However, in order to forestall any possible future compression of the cord in this zone by the growth of new bone from these raw bone surfaces, we will interpose a strip of the erector spinæ muscle. I free a strip of muscle half an inch in width and three inches in length, and swing it into the defect in the spinal column, securing it with a few tacking sutures of catgut.

We are now ready for the closure of the wound. This is effected with deep catgut and silkworm-gut tension sutures. The skin edges are approximated with horsehair. The iodin is removed with alcohol, the wound is well dusted with bismuth subiodid, and a large, moist, alcohol gauze dressing is applied, held in place with a roller bandage. The patient will be made to lie on his side only for a while.

The tendency of every tuberculoma is to heal. This boy has been on tuberculin treatment and was thoroughly prepared for this operation. We will continue the tuberculin and in a few days he will be allowed to lie flat on his back. The paralysis of motion should gradually disappear because the compression of the cord has been removed and the possibility of transmission of motor impulses restored. Remember the axons in the cord have not been destroyed. If they were, the paralysis would be permanent. However, it could disappear even without an operation. I had one case, a young woman, about twenty years of age, who had paraplegia, the result of a spinal column tuberculosis. She was in an emaciated condition at the time, and had bed-sores, so that we did not dare to do any kind of an operation. If you operate in the presence of bed-sores, you may get an infection that will cause a lot of trouble, and even the death of the patient. We started in on the tuberculin treatment. She stayed in the hospital for five weeks, then she went home. I gave her doctor full instructions about the administration of the tuberculin. He kept it up for about four weeks, then discontinued its use, because he did not believe it was doing any good. The girl's mother came to me in great distress because of the doctor's attitude. I placed myself in communication with the doctor and told him how important it was that the patient should be kept on the tuberculin treatment. He promised he would continue the injections and I must say for him that he did so, following my instructions to the letter. She improved rapidly, and after eighteen months was completely cured. The paraplegia had entirely disappeared. Why? Because the granuloma had become absorbed The tuberculin did that. [For details of tuberculin treatment see October (1913) CLINICS, and page 941 of this issue.]

I feel that, in the management of these cases from the very beginning, we are going to do our real work; it must be prophylactic work. We have not treated spinal column tuberculosis in the thorough manner that we do tuberculosis in other portions of the body. We have put on fixation casts. You might just as well put a postage-stamp on the spine, as far as getting any relief from the pressure on the cord is concerned. If you are going to relieve the pressure on the cord, the plan of treatment must be to put the patient in bed, flat on the back, so as to take the tension off all the muscles of the body which they are on all the time we are in the erect position. You do not need to do anything else to relieve the pressure due to the angulation, which is caused by the tension of the muscles drawing the diseased vertebræ together until their bodies collapse. The ambulatory treatment of tuberculosis is all right if it relieves the cord of the pressure from the infected focus. That is all. The ambulatory treatment of tuberculosis of a joint by means of a cast does not relieve the interarticular pressure at all. It merely fixes the joint, immobilizes it, and puts it at rest. We must treat these cases in a practical way, and with due regard for the pathology present. Relieve pressure and relieve tension; prevent mixed infection, check the disease, and bring about the absorption of the disease products. When you open an abscess or phlegmon of the thigh, it is done not so much for the purpose of taking out the pus as it is to relieve the tension on the pus products. It is the pressure which renders the wall of the abscess ischemic and prevents the leukocytes from absorbing the disease-products. That is it. When we treat these conditions in a rational way, then we will get results.

In this class of cases it is important to put the patient at once to bed and keep him in bed; second, give him plenty of fresh air;

third, proper feeding. Feeding is important. Fourth, we believe that one of the most important factors in producing a cure is the proper administration of tuberculin.

It is my belief that this patient will commence to progress rapidly to a cure and get motion in his legs. He still has control of his bladder and rectum. I am sure that there is no degeneration of his cord, because a compression that would produce a degeneration of the cord would also have stopped the reflexes which are still present. He would have lost control of the bladder and rectum.

A decompression of the cord, I believe, has a greater value than a decompression of the brain, as far as the practical application is concerned. Like decompression of the brain, it must always be done before degeneration of tissue starts or the operation has no effect. Then, you would naturally ask the question, Supposing that a segment of the cord had been destroyed by trauma or pressure, why would it not regenerate? Because the axons in the spinal cord have no neurilemma, and therefore they are incapable of regeneration. The same is true of the optic nerve and of all the nerves of special sense. If there is increased intracranial pressure from brain tumor and you wait until the patient is totally blind, then his optic nerve is degenerated, and restoration of sight is impossible, because the optic nerve axons have no neurilemma and regeneration of the destroyed axons cannot occur. Restoration of function in the spinal cord is possible only when transmission has been suspended and no degeneration of axons has taken place.

Let the record show that there was a compression of the cord from in front at the fourth dorsal vertebra; that the spinous processes and laminæ of the fourth, fifth, and sixth dorsal vertebræ were removed; that the hemorrhage from the bone was excessive and was stopped with a wax plug; that at the completion of the operation there was an inversion made of a strip of the left erector spinæ muscle, placing this between the ends of the divided laminæ so as to fill that space with a material that will give to the pressure and permit the cord to expand backward; that the cord did not appear to be softened at that point; that there was quite a lateral displacement; that the junction of the third and fourth vertebræ was loose and soft when we began to crush the laminæ, and the fourth moved easily on the application of forceps to the bone; that at the close of the operation, when we were closing up, the hemorrhage had ceased entirely; that the soft parts were united in the usual way with catgut and silkworm-gut and the skin with horsehair; that the cord was not traumatized nor the dura opened in the operation; that the sponges were all removed, none of the smaller sponges having been inserted. If that wax plug had not stopped that bleeding from the bone, then I would have been compelled to press into the cancellated tissue of the bone one of the small Cushing sponges tied to a string. I did not have to do that.

In connection with this case let me refer briefly to another case to contrast to this. The patient was a young man, I should say about thirty-five. He had complained of some pain shooting forward in his body for some weeks. He then complained of some pain in his spine whenever he bent forward; then he began to have symptoms of compression of the cord, slowly increasing at first, then rapidly increasing, so that in three weeks he had a complete paraplegia. I saw him in consultation. There was no history of syphilis, but the skiagram showed an apparently solid tumor of the spine in the middorsal region. The only conclusion we could come to in that case was that it was an arterial tumor, and that pressure on the cord was being produced by a tumor filled with blood which gave a shadow of the same density as the aorta. The conclusion I finally arrived at was that it was an aneurysmal sarcoma of the body of the vertebra, and that an operation was not advisable. In this the neurologist did not concur. I argued that if we operated in that case, the chances were all against us—that the patient might die on the table if we cut into this aneurysmal sarcoma. He would bleed to death. The doctor said, "but he has no chance unless you operate and there is the possibility that you are in error in your diagnosis. I think he should have the advantage of a decompression." I very reluctantly consented to the operation. Before taking out the spinous processes, on displacing the muscles laterally, as you saw me do today, I found a mass of granulation tissue coming through between two of the laminæ. I lifted that up and there was a gush of blood. We compressed the tumor with sponges until the wound could be closed. The microscopic diagnosis of a small portion of tissue removed showed at this time aneurysmal sarcoma, confirming the clinical diagnosis. See what would have happened if I had taken out the laminæ! You could not have checked the hemorrhage by sitting on it. We were able to check it until we got in the sutures. [For a full report of this case see CLINICS, August, 1913.] He had a little elevation of temperature all of the time, which is so common with rapidly growing sarcomata in young people—even a temperature as high as 103°–104°. One of the first cases of this kind I saw was when I was an intern in the Cook County Hospital. The patient had a temperature of 104.5° F. following a trauma—an acute trauma to the leg. It came on suddenly, as in an acute infective osteomyelitis. In a few days he had a pulsating tumor and we amputated through the thigh. Examination of the specimen showed that it was an acute aneurysmal sarcoma. I cite that case to accentuate the fact that elevation of temperature can be present with a sarcoma as well as with an infection, and unless one is careful he will open one of these tumors for the purpose of evacuating pus causing the patient to bleed to death.

SUBCUTANEOUS ABSCESS FOLLOWING TUBERCULOSIS OF THE SPINE. ASPIRATION AND INJECTION OF FORMALIN AND GLYCERIN SOLUTION

HISTORY

Patient, female, aged fifty-three, entered hospital complaining of weakness, loss of weight, and pain in the lumbar region on attempted flexion of body at waist. She has been in bed continuously the past three weeks, and most of the time since February, 1913. She says she has a drawing pain in the back when she attempts to walk or raise her arms above her head. The trouble began early in January, 1913, following a cold with cough that had lasted two months. She first noticed a sore and tender spot just to the left of the spine in the lumbar region. The soreness came on gradually. There was no swelling. Then she had pain, which quickly increased in severity in a few days so that she went to bed. She could not sleep because of the pain. At this time she was treated for "rheumatism." She did not have chills, and does not think that she had a temperature. After two or three weeks the pain shifted to the right side of the spine. About this time she first noticed a deformity of the spine in the lumbar region and a hard tumor over the spine. This tumor was not tender to pressure nor painful, and was situated above the tender and painful areas to the side of the spine. About the middle of February an abscess formed to the right of the spine in the lumbar region. This has been aspirated twice and pus evacuated, but it filled up again, has grown larger, and is getting more tender and sore. She never had a cough before the "cold" which preceded the present trouble, but six or eight years ago she had profuse night-sweats for a year or two. She has never lost much weight, but has been getting weak and had malaise during the past two years. She had typhoid nine years ago. Was in bed five weeks. Family history is negative to tuberculosis and carcinoma.

Examination (July 8, 1913) showed an abscess to the right of the spine in the lower dorsal region. It is a tubercular abscess following a tuberculosis of the spine.

COMMENTS AND OPERATION

Dr. Murphy (July 10, 1913): One does not need any more information than is given in that history to tell the story of that "rheumatism" in the back. It is the usual story. When a patient complains of pain in the back an examination is not usually made of the spine. The case is treated as one of lumbago or neuralgia. You can, as a rule, make a diagnosis in these cases from the fact that there is early fixation of the spine and the patient complains of pain increased by motion. These are the first symptoms. Every year we have a crop of cases of spondylitis. That crop comes to us from the rural districts within a radius of one hundred miles from Chicago. Usually they begin to come about the fifteenth of November, and they continue to come from that time until March. They come with various degrees of spinal fixation, but all complaining of pain in the back or of referred pain to the pyloric, gall-bladder, or appendix zones.

We have in the hospital now two cases of spondylitis of the occupational origin type. The men patients are farmers, who work during the fall in a stooping position, husking corn and digging potatoes, etc. They are subject to the nasal and bronchial infections prevalent during that season of the year; then they get wet and work all day in the stooping position; they commence to complain of pain in the back. They usually have some type of infection which is metastasized to the occupational overcompressed zone of the spine.

These cases come in every year and in increasing numbers. Their number is rather appalling. Whether we get them from the fact that our office is the sifting-place or whether this experience is in common with that of other men I do not know. A man speaks of the cases that come to him, but that is not always a fair basis on which to make an estimate when you speak of percentages. However, we speak of percentages as the cases come to us. These are cases of metastatic spondylitis and not lumbago.

This is a case of tuberculosis of the spine with abscess formation. She has a kyphosis in the lower dorsal region. That tells the whole story. First, there is the history of having felt far from well for a long time before any specific symptoms were manifested, which is true of tuberculosis anywhere in the body. Remember that tuberculosis of itself never kills. Tuberculosis threatens life only when a mixed infection takes place. The tendency of tuberculosis in every location in the body, except in intestinal ulcer, is always to heal. The focus is either surrounded by connective tissue—encapsulated—or it heals by cicatrization. An intestinal ulcer of tuberculous origin never heals of itself, because of the constant irritation to which it is exposed by the intestinal contents passing over it. These spinal cases very frequently are caused by the simple tubercular infection, not mixed infection. That infection always takes place in the body of the vertebra, never in the processes. It begins either in the anterior, lateral, or posterior part of the body of the vertebra. The granuloma or tubercle forms, the center of the mass breaks down, and if the lesion is in the anterior portion of the body of the vertebra, a kyphosis is formed as soon as the vertebra collapses. That collapse causes an increased intra-articular pressure on the posterior spinal nerve-roots and the patient then has pain, which increases in severity until the spine becomes fixed or immobilized as a result of nature's effort to heal the disease. That gives us the indication for treatment in these cases. When the tuberculous lesion begins in the posterior portion of the vertebral body, a lordosis occurs. You can always get a clean-cut history of trauma in these cases. I remember a patient who came here from Ireland with a pain in his back. He gave this history. He went into the barn one night without a light. The children had tied a rope across the door. He caught his feet on it, fell forward, and struck the ground forcibly on his elbows, his heels flying upward so that he arched his back. What happened to his spine? The spine doubled backward and the pressure was thrown on to the posterior portion of the body of the vertebræ. He went along for months with pain in his back, and finally he commenced to develop a lordosis. A tuberculin test was made and gave a positive reaction. He developed an abscess—just like this woman did. I cite that case merely to emphasize the close relationship between the trauma and the localization of the tuberculosis in the spine. When the vertebral body weakens behind, the spinous processes are jammed together and overlap as the body collapses. Kyphosis is caused by an absorption of the vertebral bodies in front, the posterior portion of the body sustaining the support, causing the spinous processes to separate and become prominent as this woman's are.

The tumor in this case is distinctly fluctuating. We will aspirate it. It extends from the ninth to the eleventh dorsal vertebra, with the center of the swelling to the right of the body of the tenth vertebra. That is the part of the spine which is most commonly traumatized. It is the position in which one usually finds the lesion. She has had a tuberculosis in the anterior portion of the body, but the tuberculosis, instead of progressing along down in front of the spine and forming a psoas abscess, extended backward and formed this subdermal lumbar abscess.

There is a well-recognized law in tuberculosis that you must never open a cold abscess that communicates with a tuberculous focus unless you can go to the bottom of it and clean out the primary focus. You must not open a tuberculous abscess as a matter of election, because the moment you do there is danger of admitting a more virulent and dangerous type of infection, converting a simple tuberculous into a virulent mixed infection. Circumscribed abscesses should never be disturbed to the extent that there is danger of causing a general infection by overpowering the local encapsulating tissue resistance, because then absorption of the septic products takes place, and the patient gets chills, fever, night-sweats, and other evidences of a spread of the infection throughout the body.

In this case it is very desirable that this woman should not get a mixed infection. We must endeavor to stimulate the formation of connective tissue in the wall of the abscess, so that if it is opened subsequently the lymph-spaces in the immediate neighborhood will be coffer-dammed and absorption of septic products cannot occur. How do you coffer-dam the tissues? By injecting into the abscess any material that will stimulate the production of connective tissue. Iodoform is one of these. Turpentine does not stimulate the formation of connective tissue to a great degree, but it causes a local polynuclear leukocytosis. Formalin is better than either iodoform or turpentine. Formalin produces a polynuclear leukocytosis and also stimulates the formation of connective tissue. It most effectively coffer-dams the lymph-spaces and prevents absorption of the infective products.

Our desire is to aspirate this abscess first, withdrawing as much pus as possible. Aspirate it not on the line of gravity, where a permanent sinus might be formed, but where there will be the least pressure—at the upper outer angle. The abscess will probably finally open of itself. If we can postpone this opening until such time as the neighboring tissue is thoroughly infiltrated with polynuclear leukocytes, and the lymph-spaces are coffer-dammed, then the patient will not be exposed to the danger of a mixed infection or an absorption of septic products that she would be if you open the abscess now.

If this tuberculous process had involved the lumbar vertebræ in place of the dorsal vertebræ, then this woman would have had a psoas abscess in place of a lumbar abscess. We sterilize the skin by painting it with iodin, and then we freeze the site to be punctured with ethyl chlorid. We will use a large blunt needle. There is the pus, showing that we are in the abscess cavity. We have to use a screw syringe to evacuate this material. It is very thick and contains many flocculi. We have withdrawn about two ounces of pus and débris. We leave the needle in the abscess, unscrew the barrel of the syringe, and replace it by one filled with a 2 per cent. solution of formalin in glycerin at least twenty-four hours old, then inject it, withdraw the needle, and seal the opening with collodion. This abscess cavity will fill rather rapidly now with fluid on account of the stimulation of the formalin and glycerin. Remember that the formalin and glycerin go to the bottom of the cavity because it is heavier in specific gravity than the fluid.

[The patient reacted well to the injection. The abscess was aspirated again in a week, and in three weeks only a little fluid was removed at the last aspiration. The pain and tenderness disappeared, and the patient was very comfortable. She left the hospital two days after the last aspiration. The formalin and glycerin injection was not repeated.—Ed.]

UNDESCENDED TESTICLE IN INGUINAL CANAL

HISTORY

Patient, male, aged forty-four, enters hospital on account of an undescended right testicle and a tumor mass which appears in the right inguinal region after straining or working hard or after walking for some distance, especially when not wearing a truss. It is the size of a fist and soft. This patient has been wearing a truss as long as he can remember. When five or six years of age he received an injury in the right inguinal region, being struck by a stone. When Christian Science came into being in the eighties he became a convert to it in the belief that the hernia would be corrected. He discarded his truss, played football, and took part in other games. Always, however, the tumor mass would present itself in the groin. After going to bed he would replace the mass by manipulation, and at times it receded without such aid. Fourteen years ago he began to wear the truss again and he has been wearing it ever since. He claims that at times the testicle slips out and appears at the external ring. He suffers pain then on account of the pressure of the truss on the testis. The mass has never become strangulated.

At the time of examination there was found an enlarged right external ring, with a protrusion of some abdominal contents on coughing. No bulging was otherwise apparent. The testicle was not palpable either at the inguinal ring or in the scrotum. The left testicle is down in the scrotum. Patient had gonorrhea twenty years ago. Denies lues. Family history negative, except that mother died of carcinoma of the breast.

COMMENTS AND OPERATION

Dr. Murphy (operation September 22, 1913): The intern states that the tumor is of the size of a fist. I do not think that is correct. The impression I got from the patient was that it was about the size of a testicle. If it were the size of a fist, it would be a large scrotal hernia. The treatment would be an entirely different proposition if this man has a sac the size of a fist down in the scrotum from what it would be if it were a sac of peritoneum which comes down to or just below the external inguinal canal opening. That is why I want to correct that impression now. What is the matter with him?

INTERN: He has an undescended right testicle with an enlarged external ring. At times a portion of the hernial sac comes down, filled probably with intestines.

DR. MURPHY: What makes you say that last? What makes you say "filled probably with intestines"?

INTERN: Because the mass appears especially when he has taken an exceptional degree of exercise or after long walks.

DR. MURPHY: Yes.

INTERN: Or a portion of omentum——

DR. MURPHY: Yes, what else?

INTERN: Or the bladder might be in the sac.

DR. MURPHY: Yes. What about the testicle coming down first? The testicle is attached to the peritoneum through what? The gubernaculum testis; therefore, if the sac comes down, what should come down with it? The testicle, if it is attached. Yes. Why do you say that it is the intestine and not the testicle?

INTERN: I would say both came down.

DR. MURPHY: Yes, but why do you say both? Why is it not only one—the testicle? If the mass were as large as you say, then he would have discomfort always. If only as large as I say, then he would have to do only with the testicle. He gets a sickening pain when it is down. He says when he palpates it he feels first the testicle.

That brings up the subject of ectopia of the testicle, or undescended testicle. When the testicle first descends in the fetus, where are the regular places into which it descends? Before we go into that, let us first ask ourselves, "What is the scrotum?" The scrotum is a sac made up of skin and dartos. Normally, the inner portion of the dartos is lined by peritoneum. The

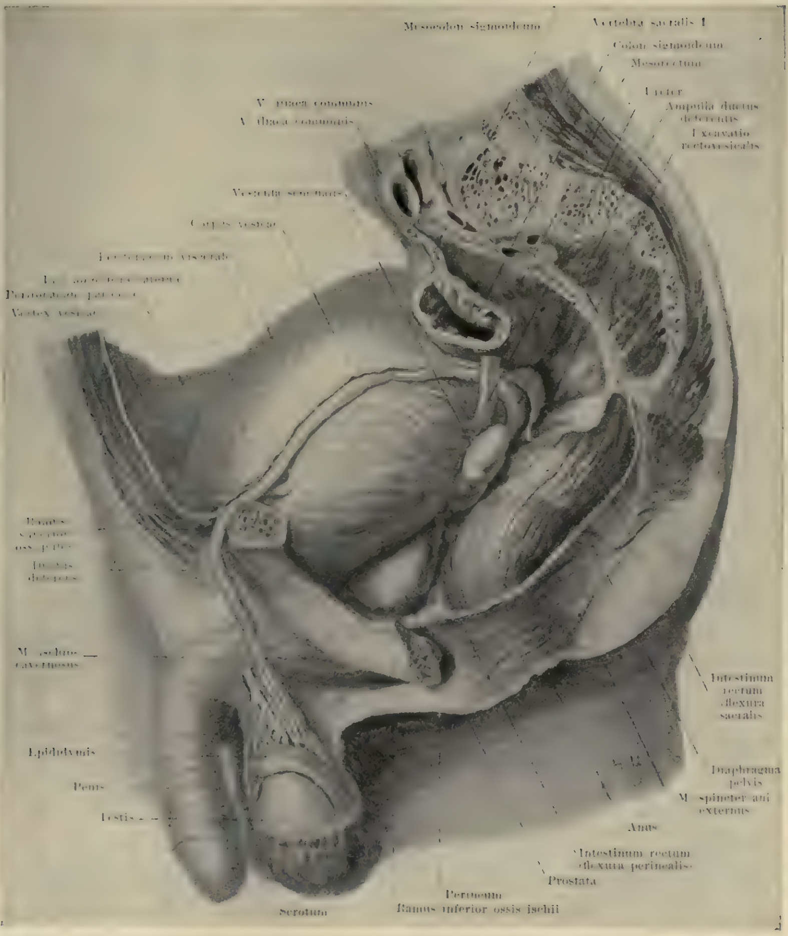


Fig. 255.—Male pelvic organs after removal of the left pelvic wall. The urinary bladder is fully distended. (After Spalteholz.)

scrotum is the continuation of all the layers of the abdominal wall with the muscle-fibers almost entirely absent. That is all it is. There is, therefore, in the dartos a representative of all the layers of the abdominal wall coming down from the external inguinal opening. About two years ago we had a very striking case, one that had been all over the world for examination. It was a case in which the sac, as far as the skin was concerned, was perfectly normal, but there was no dartos tissue in the scrotum. In other words, there was no descent of the representatives of the other abdominal layers down into the scrotum. That is one of the most unpleasant conditions in connection with cryptorchism. That boy had his dartos extending across the symphysis, above the penis, making a large pouch over the symphysis. Both testicles were in the abdomen. When there is a failure of descent of the dartos, naturally the testicle does not come down. But the dartos may come out in another direction, *i. e.*, upward, on the anterior abdominal wall, and the testicle may be carried in the sac to that position and the scrotal skin remain empty.

The descent of the testis is controlled by the gubernaculum testis, which is attached to the lower pole of the testis, the globus minor of the epididymis, the cecum, and the skin at the bottom of the scrotum passes over to the perineum and symphysis pubis and the anterior spine of the ilium. The arrest of the testis at any point constitutes the imperfectly descending testis. Its departure from the normal direction into that of some of the nonscrotal fibers of the gubernaculum constitutes the ectopic testis. The testis may, therefore, be situated in the abdomen, in the inguinal canal, just outside the external ring, or in the perineum, over the symphysis pubes, or near the anterior superior iliac spine.

The cause of the imperfect descent is unknown. Such a testis is nearly always undeveloped and functionless as far as the secretion of spermatozoa is concerned. In all cases of this kind the processus vaginalis is found in the scrotum, but the upper end may not become shut off from the general peritoneal cavity, and the patient always has a potential or an actual inguinal hernia.

Fluid may collect in the peritoneal pouch and form a congenital hydrocele.

There is still another type. We have one example of it in the case of a man who has been coming to our office for over twenty years. His testicle comes out directly through the internal ring and passes on to the anterior abdominal wall. There is a dartos on the anterior abdominal wall, which you can feel distinctly, and the testicle is in it.

Dr. Wells, the statement was made in the history that this tumor came out as large as a fist. Will you kindly tell us how large it appeared to you? I took exception to the statement because I felt that it was wrong.

Dr. Edward F. Wells: There was an external protrusion, but it was not excessive at any time. I would say that it was the size of a lemon. At one time, some years ago, it was about that size. That is probably twelve years ago. Since then I have never seen it of that size. He has worn a truss.

Dr. Murphy: Did it ever come down into the scrotum?

Dr. Wells: No, sir; distinctly no. At least I have not seen it so. I think it is altogether in the canal.

Dr. Murphy: To go on with the failures of descent, the testicle may be arrested in any part of the tract from the position of its primary formation at the lower pole of the kidney down to the end of the scrotum. An undescended testicle may start from the kidney and proceed only a few inches toward the inguinal canal. It may start from the kidney, and in the malformations that occur it may go down into the pelvis, and attach itself to the end or side of an anomalous tube. I operated on a patient, a male, about two years ago, who had a well-developed uterus and tubes, a fairly well-developed vagina, no scrotum, and testicles within the abdomen where the ovaries should be. One of these testicles was sarcomatous, so I removed it. The other one was normal and we permitted it to remain in the abdomen. I have not heard from the patient since, so I do not know the ultimate outcome of the case. The testicles were free in the peritoneal cavity, only attached near the fimbriated end of the tube on the posterior surface of the rudimentary broad ligament. Microscopic examination of the removed testicle showed it was a round-cell sarcoma of a testicle.

The diagnosis of undescended testis is usually easy. It is made by finding the scrotum empty, and, if the gland is not situated intra-abdominally, by feeling it in its abnormal position. In imperfectly developed or undescended testes the gland cannot be made by manipulation to reach the bottom of the scrotum, thus differentiating it from the mobile testis, which can readily be pushed down into its proper place.

Operation is indicated in all cases, except in those in which the testis is in the abdomen. The gland may be either fixed in the scrotum, removed, or returned to the abdomen.

Now we locate the anterosuperior iliac spine and the symphysis. We make the incision parallel with but about one inch above Poupart's ligament, extending from the anterior iliac spine to just below the external inguinal ring. It is the usual incision in operating for a hernia. We always make that incision high on the abdomen, and never on the inguinal canal or tract. We divide the skin and fascia down to the aponeurosis of the external oblique muscle. Retracting the wound margins, we come down at once on the sac of the hernia, and in it is the testis just outside of the internal inguinal ring. The tissues are not disturbed or covered with blood. You can see them clearly.

We proceed with this operation the same as we do for a hernia. [For a full description of this technic see CLINICS, October (1913).] Here, you see, is the continuation of the sac down in the direction of the scrotum. There is the continuation of the canal. I am peeling this off, though the testicle is visible right there in the sac. See! that looks like the tunica albuginea, in the continuation of the peritoneum downward. There is the testicle right in the sac. That is as far as it goes down, so that at no time did it go down into the true scrotum. There is the continuation of the dartos tissue downward. Let us examine and see what kind of a testicle we have to deal with. It is normal, except the epididymis is only attached to the upper pole. See the veins which come down on the side! The spermatic artery comes off from the internal iliac or from the superior vesical and is quite long in this case.

While that testicle is malformed as far as the epididymis is concerned, it is not aplasic, therefore I will save the testicle. An aplasic testicle is always the source of great danger. A large percentage become sarcomatous and a smaller percentage become carcinomatous, the sarcoma being the one that occurs in the early periods of life and the carcinoma in the later periods of life.

Now we have the testicle free. We will handle it on the principles laid down by Dr. A. D. Bevan in a very able article presented on this subject some years ago. Now I have brought up the most dependent portion of this testicle. I will make a pocket for it in the scrotum. There is the dartos tissue shining here. These tissues separate very easily. The testis can now be drawn into the scrotum and we will fix it to the connective tissue of the scrotum with a few catgut stitches, and two horsehair sutures passed from without through the scrotum and into the connective tissue at the lower pole of the testis secure it in position.

There was no normal inguinal canal present here, so that we brought out the testicle direct. That gives a short route for the vas and for the spermatic vessels. If we had attempted to bring it through the upper portion of the canal, we would have a longer route for the spermatic vessels and necessarily put greater tension on them. With two chromicized catgut sutures I will unite the conjoined tendon and the aponeurosis of the external oblique to Poupart's ligament, the same as you do in the Andrews operation for hernia if you are doing it without this testicle proposition at all.

I make a canopy of the canal by putting Poupart's ligament over it. Here all the knots come on the outer side. That gives us a good support and utilizes the aponeurosis of the external oblique twice—once as the true aponeurosis and once as its continuation, as represented in Poupart's ligament.

In aplasia of the testicle one has a very serious proposition to consider as to what it is best to do. When there is a small testicle proper, that is, when the glandular structure of the testicle is aplasic, that is the most serious condition, because it is embryonically defective, therefore much more liable to malignant disease. In this case the testicle was apparently normal, but there was a failure of attachment to it of the epididymis. When you follow the seminal tracts out it is a very simple construction. First, there are the seminal tubes passing into the longitudinal tubes that pass into the rete testis or hilum of the testes; then there are the vasa afferentia going into the numerous vessels that make up the epididymis; then you have the formation of the vas, which is a continuation of the conducting tube.

The chances of a recurrence of the hernia are greater in this case than in the ordinary case where the testicle is up and out of harm's way. The reason is because you have to leave more tissue in the cord than one necessarily does when the hernia is of the usual kind.

Let the record show that Sister reports the sponges counted and correct. Let the record further show that the testicle was situated just outside of the internal inguinal ring.

Now I will close the connective tissue up and down on the level, so as to prevent any portion of the testicle from returning into this field and becoming adherent and fixed, and also avoiding a dead space. In freeing the testicle to bring it down in a case where it is situated higher up, one and one-half to two inches from where it is here, it is then necessary to make a free opening of the peritoneum and to free the vessels behind the peritoneum so as to get the tension off those vessels, the veins, and spermatic artery. Remember, the vessels of the vas are usually ample to nourish the testicle.

In operating for varicose veins is it possible to ligate everything in the cord and have the testicle live? Yes, but I do not want you to do it, because sometimes, where you have ligated all the veins, you get, following that, a descending thrombosis clear down into the veins of the testicle. You get a gangrene—an aseptic necrosis with absorption finally that comes from the thrombosis. It does not come from the primary ischemia—it comes from the thrombophlebitis caused by the ligation. It is not common, but if you will look through the literature, you will find quite a number of cases of that kind recorded.

Let the record further show that the testicle was normal so far as the tunica albuginea, the tunica vaginalis, and rete testis were concerned; that the epididymis was not attached normally; that the globus major was perfectly free; that as there was no aplasia of the testicle proper, the testicle was not removed.

A retained testis is usually imperfectly developed, and it has been claimed that this was the cause of the undescent. On the other hand, the descent of the testis may be considerably interfered with by a short or non-patent processus vaginalis, as well as by adhesions between it and the tunica vaginalis. It is not always possible to bring the testis down into the scrotum and to hold it there. Numerous attempts have been made to do this, such as fixing it to some neighboring part of the thigh, stitching it to the scrotum or to the other testicle, pushing the testis out through a slit in the bottom of the scrotum, and stitching it to the thigh. De Beule stitched the scrotal wound as well as the edges of the incision in the thigh so as to cover over the testicle.

Lanz's method is more simple. He passes a thread through the tunica albuginea of the lower pole of the testicle, brings it out through the scrotum, and fixes it. Kocher advises to divide all the attachments in the neighborhood of the spermatic cord because otherwise the circulation in the cord is endangered by the tension on these structures. Kocher fixes a thread to the lower pole of the testis, brings it out through a small opening in the bottom of the scrotum, pulls down the scrotum on the thigh, and fixes it there with the same thread, which has been left long purposely. In this way absolutely no wound is left open.

CHOLELITHIASIS; STONES IN COMMON DUCT, WITH INTENSE JAUNDICE

HISTORY

Patient, female, aged forty-five, enters hospital on account of attacks of pain in the epigastric region, followed by nausea and vomiting, and an extreme jaundice that is increasing in degree. For the past ten years she has had attacks of a dull, heavy pain in the epigastric region, with tenderness on pressure over this zone. About one to one and a half hours after the onset of the pain the patient would become nauseated and then vomit, which relieved her of the pain for a while. The pain had no relation to eating. It was not initiated by eating nor was it relieved by eating. The attacks lasted two or three days. She never had any chill, fever, or jaundice at that time.

Since Easter-time, 1913, the patient, who had been free from these attacks for one year, has had almost constant pain and tenderness in the epigastrium, with occasional attacks of nausea and vomiting. Saturday, April 12, 1913, she had a dull, heavy pain in the epigastrium, with tenderness over this region. Two or three hours after the pain started she became nauseated and vomited. Vomitus contained mucus and bile. Pain and tenderness continued. Wednesday, April 16, she began to become yellow. She has had no chill or temperature elevation. The jaundice has increased in degree, until now she is of a bronze color. She had pneumonia twenty-four years ago. Never had typhoid. Family history negative. Personal history otherwise negative.

COMMENTS AND OPERATION

Dr. Murphy (operation June 6, 1913): What was her condition on admission?

Intern: She was very much jaundiced.

Dr. Murphy: Yes, jaundiced in the superlative degree. She had a colic how long preceding her admission?

Intern: April 12th. She came in on the eighteenth.

Dr. Murphy: When was it that she first noticed that she was jaundiced?

Intern: On the sixteenth.

Dr. Murphy: She began to get yellow four days after the onset of the pain. The jaundice became more and more intense. Finally she began to have hemorrhages the result of the intense icterus. Her coagulation period was six minutes. Blood came from the bowels, from the nose, gums, and bronchi, and she had a hemorrhagic purpura. It looked as though she was going to die from the acute anemia. She did not bleed from the kidneys. We secured some coagulose and gave her two doses, twenty-four hours apart, as recommended to me personally by Dr. Stockton, of Buffalo. That seemed to aid in stopping the hemorrhage. She remained jaundiced for about ten days or two weeks, then she suddenly got well. She has been in the hospital for six weeks; during this time she has had thyroid gland and calcium salts to lower her coagulation period and trying to get her in condition to justify an operation. We believe that a stone was passing out of the common duct either back into the gall-bladder or on into the bowel. We are operating today with the hope of not finding a stone in the common duct. What is her coagulation period?

Intern: Two minutes, ten seconds.

Dr. Murphy: That, you see, is within the average range. We hope and believe that we will not find a stone in the common duct, and, I assure you, we do not want to.

This is the only case of this kind I ever saw that recovered. If we had operated on this woman at the time she was jaundiced and had her hemorrhages she would have died. This was purely a matter of judgment. We know that in these superlative jaundice and septic cases, if you do the slightest operation, the patient will die. (See Kehr's early reports.) We tided her over and gave her by proctoclysis a 0.6 per cent. solution of calcium salt to help to get her into a condition so that the blood would coagulate. Unless you have plenty of calcium in the blood you do not get normal coagulation. That is why we give salts of calcium in these cases, as recommended in my paper in 1892.

I will make the incision on the outer margin of the right rectus muscle. This woman is very fat, and we will have to make a longer incision than we usually do in order to get a good exposure.

We incise the anterior layer of the rectus aponeurosis and displace the muscle inward. The peritoneum is incised in the usual manner—on the incline between two tissue forceps which grasp the peritoneum. If any intestines are in the grasp of the forceps or adherent to the anterior wall or peritoneum, they are less likely to be cut.

I am going to palpate the gall-bladder. It is thickened and whiter than normal, but it does not contain any stone. Extending my examination to the common duct I can feel a stone of some size—as large as a pecan-nut. I want to see if I can work the stone back out of the common duct into the cystic duct and then back into the gall-bladder. If it goes back into the gall-bladder, it will be very fine. I cannot budge it; therefore I am forced to open the common duct in order to remove the stone. It must be removed because it will not move voluntarily in either direction. One dislikes very much to open the common duct in a patient as fat as this woman is. First we will expose the duct well. It is a white tube, about the size of a lead-pencil. Then we will aspirate it to make sure that we have the right structure. We grasp the duct very carefully with tissue forceps and then incise it, making a longitudinal incision long enough to extract the stone. By gently milking the duct and using a small spoon, we have removed this stone. There is another stone here in the duct. It seems to be lodged more firmly than the first. It is in a diverticulum of the duct. That stone would never cause jaundice because it does not obstruct the lumen of the duct. The other stone completely closed the duct, so that the bile was dammed back into the liver and entered the circulation, this resulting in the intense jaundice. We have removed this second stone. It is smaller than the first. Both are smooth. We will drain this duct and also the gall-bladder and the hepaticoduodenal fossa.

We place a small rubber tube into the common duct and extend it up into the dilated hepatic duct (this is what Kehr calls drainage of the hepatic duct), and larger ones into the fossa and the gall-bladder. The tube in the gall-bladder is placed in the usual manner. Two purse-string sutures of plain fine catgut are placed in the dome of the gall-bladder one-half inch apart. The tube is inserted into the viscus in the center of the top suture. This is drawn taut around the tube and is fastened to it by one or two stitches made with the same needle used in passing the purse-string suture, then making the end of the gall-bladder fast to the tube. The tube is pushed down into the gall-bladder so as to invert the top purse-string. The second purse-string is tied and secured to the tube in the same manner as the first. Thus the tube is made a part of the suture and it is secured in place with an entropion of the cut edge of the gall-bladder.

Let the record show that the smallest tube is in the duct; that the large tube is in the free peritoneal cavity; that the middle-sized tube is in the gall-bladder. There was practically no bleeding here during the operation. The stones had passed out of the gall-bladder and through the cystic duct into the common duct. I tried to get them back into the gall-bladder, but it was impossible, so I decided to open the common duct, which I did; that before opening the duct, we aspirated it with a fine needle and saw that it was the duct. The first stone was removed rather easily; the second one was very difficult of removal; it was in a diverticulum.

Let the record further show that Sister reports the sponges counted and correct.

This patient came here on the eighteenth—six days after she had the first pain. She was intensely jaundiced. We thought it would cease and the stone pass into the common duct. Finally the hemorrhages began. She had reached the hemorrhagic stage. She passed blood from the bowels and from the nose. It seemed as though she was going to die. We felt that it would be fatal to operate on her at that time, so we just tided her over. While I was in Buffalo recently talking with Dr. Stockton we discussed the value of coagulose in stopping hemorrhage. His confidence impressed me very much. Therefore, when this patient came to us in the condition she was in, we immediately gave her two doses of coagulose. It stopped the hemorrhagic condition. Gradually she began to retain a little on her stomach. She had been having emesis as a result of the intense icteric condition. Finally, the procteal calcium salines were retained.

Notwithstanding the jaundice which had existed since the sixteenth of April,—almost two months,—you will note that we had very little bleeding. This shows that the coagulation period of the blood returns rather rapidly to normal when the jaundice has been of short duration. Just what effect coagulose has on the blood no one knows. Finally, the coagulation test was made, and we found that it was two minutes and ten seconds. Fearing that she would get another attack of jaundice, we decided to operate earlier than we had originally intended. This is the first case of icteric hemorrhage of this severity that I ever saw recover. We will give her calcium proctoclysis for a few days.

[This patient progressed well. The salines were discontinued on the fourth day. The fossa drain was removed on the sixth day, the common duct drain on the ninth day, and the gallbladder drain on the seventeenth day. Every trace of jaundice had disappeared. The wound closed quickly. The patient left the hospital six weeks after the operation in perfect health.—Ed.]

TALK ON CHOLELITHIASIS AND CHOLECYSTITIS

Dr. Murphy: I want to talk to you this morning on cholelithiasis and cholecystitis, and point out the relation which the pathology has to the clinical course of the disease. Interpreting signs and symptoms into pathologic entities is the most difficult proposition in medicine. When I was a student in Berlin in 1883 we had a course of lectures by Schroeder, then professor of gynecology in the Frauen-Klinik in Berlin. They had a system over there which was admirable. They had a number of hospital externs called *Practikanten*, who worked only at night and responded to obstetric and gynecic night calls from any part of the city. In the morning these men came into the clinic, and each man gave the history of his particular case which he had charge of during the night and told us what he did in the way of treatment. He also presented the after-birth, fetus, abortion result, etc., which he captured in the night. I learned more in that clinic than I did in any other clinic I ever attended, because Schroeder went into the details of the history, discussed the pathologic findings, and gave us the result. That is my purpose in bringing before you the histories of two cases we had a few days ago. In the history of the first case I want you to note the long duration of the condition—from the first attack until the present time. This man has had no relief from the first symptoms to the final symptoms which caused him to come here. The final symptoms were the serious symptoms which so greatly hazarded his life. All of this was avoidable, if the warnings of nature in the form of pain in the right hypochondrium and the other symptoms had been recognized. Let us listen to the history of the first case.

HISTORY

Patient, male, aged sixty-nine, enters hospital on account of attacks of acute colicky pain in the right hypochondrium, followed by nausea and vomiting. He had typhoid thirty-five years ago (1878) and was sick seven weeks. Twenty years ago he had an attack of acute colicky pain in the upper part of the abdomen, more to the right than to the left of the midline. The pain continued one hour. He was not nauseated and did not vomit. One month later he had another attack of pain lasting about one or one and one-half hours. He was nauseated and vomited, first one hour after the onset of the pain, and again shortly after. He was not jaundiced. He had no further trouble until five or six years ago, when he had attacks of sharp colicky pain in the right hypochondriac region lasting one or one and one-half hours. He was nauseated and vomited, one-half to one hour after the onset of the pain. Since that time he has had numerous similar attacks of pain, lasting from one to three hours, followed one-half to one hour after the onset by nausea and vomiting, and occasionally slight jaundice, lasting one or two days. These attacks came on every month or two. March 19, 1913, he had an attack of sharp pain lasting one hour. One-half hour after the onset he was nauseated and vomited. He became jaundiced. The jaundice did not diminish in degree, but became worse. The urine became very dark, and the stools light—clay-colored. The jaundice has persisted until the present time, and the stools are still clay-colored. Since March 19th he has had three or four attacks of colicky pain with chills and high (?) fever. The patient is sore and tender in the right hypochondriac region during these attacks. He never had a chill preceding an attack or fever during or following the attack before March 19th. He has lost 15 pounds in weight during the last two months. Family history is negative to tuberculosis and carcinoma.

DR. MURPHY: He had typhoid in 1878?

INTERN: Yes.

DR. MURPHY: The most common etiologic factor of gall-stones is typhoid. The bacilli always reach the biliary passages and are excreted in the bile. The typhoid bacilli are found in the gall-bladder for a long time after the subsidence of the disease. The gall-bladder becomes infected. Remember that all gall-stones are the sequences of an infection. The infection is more commonly in the gall-bladder than in the liver or in the biliary ducts. The majority of the stones form in the gall-bladder, and 98 per cent. of the weight of all stones is cholesterin. All gall-stones are originally soft cholesterin masses. *Gall-stones are the sequences of the degeneration of the epithelial cells lining the gall-bladder* or lining some pocket in the biliary ducts. Gall-stones differ from the renal or vesical calculi in that they are the results of cholesterin formation and of the degeneration of the epithelial cells, while stones in the urinary tract nearly always, but not entirely, are the results of the precipitation of salts that are in the urine. The etiology of gall-stones is an entirely different proposition from the etiology of renal or vesical calculi. The dead epithelium in the biliary passages is the product of an infection—a chronic infection. The most common infectious agent that remains active in the gall-bladder for years and produces an exfoliation and death of the lining epithelial cells of the mucosa and the mucous gland cells of the gall-bladder is the typhoid bacillus. In the great majority of cases these stones form in the gall-bladder. How do they commence to make trouble and where do they cause trouble? (*a*) In the gall-bladder; (*b*) in the cystic duct; (*c*) in the hepatic duct; (*d*) in the common duct, and occasionally (*e*) in the intestinal tract. When they endeavor to escape from the gall-bladder or when an active infection is added to the one already present, and a severe mixed infection is the result—one which causes an extensive secretion of fluid, often of pus, into the gall-bladder. They commence to make trouble when the infective products in the gall-bladder attempt to escape by the contraction of its walls, due to the irritation, and the stones are carried forward and occlude the gall-bladder neck, the cystic duct, or the common duct. Or the gall-bladder may rupture and the infective products escape into the neighboring structures—into the duodenum, into the colon, into the stomach, and occasionally into the free peritoneal cavity, or an abscess is formed which may rupture.

The history of this patient is a fairly good description of a lesion that occurred either in the fundus or in the neck of the gall-bladder. When the stone passes out of the gall-bladder into the cystic duct, the symptomatic manifestation is the colic. That colic continues during the passage of the stone. It is important to get an accurate history in these cases. A stone may pass into the cystic duct and remain there stationary for years without causing a colic. As soon as it moves up or down in the duct the colic occurs. As long as the stone is stationary in the cystic duct or while it remains in the gall-bladder there is no colic and no jaundice.

In the order of their appearance the symptoms of gall-stone disease are pain, nausea and vomiting, local sensitiveness, elevation of temperature,—if infection be present,—jaundice if the stone is passing into the common duct and obstructing its lumen. Jaundice is usually a late manifestation of gall-stone disease, because it results only when there is an obstruction to the outflow of the bile, so that it is dammed back into the biliary passages, and from there passes out from the liver by means of the hepatic veins and into the general circulation. Jaundice occurs only in about 20 per cent. of the cases, and even with one or more stones in the common duct only in 33⅓ per cent. When severe in degree and long continued, it is caused by an impaction of a stone in the common duct. If the stone lodges in the ampulla of Vater or in a diverticulum, or if it is of the ball-valve type, which was so well described by the late Dr. Fenger, the bile passes by it and jaundice will not continue, or at most only in a mild degree.

Let us analyze this man's case. He had typhoid fourteen years before he had his first colic. He had attacks of colic for three or four years, but at long intervals; no jaundice at any time, which means that the stone that formed in the gall-bladder passed down into the cystic duct, but not through it. If there is associated an infection in the gall-bladder, the patient may have a temperature of 101°–102° F., with the gangrene of the gall-bladder, but when the stone passes out of the gall-bladder into the cystic duct and becomes arrested in the cystic duct, with infection present, he will have chills and fever, sometimes recurrent, simulating typical or atypical types of malaria. Why is it that he has chills and fever of such a severe type when the stone gets into the cystic duct? Why does he not have them when the gall-bladder is full of pus? For the very good reason that the chills and fever are not manifestations of the presence or the quantity of pus, but of the degree of absorption of the pus. The gall-bladder may be full of pus for weeks, months, or even years, and the temperature never reach 100° F. Why? Because the wall of the gall-bladder has but a sparse lymphatic network, becomes infiltrated, and absorption cannot take place. When the stone works its way out of the gall-bladder into the cystic duct, it produces great pressure and causes an erosion or ulceration of the mucosa of the cystic duct; the pus is absorbed and the patient has chills and fever because here there is a rich lymphatic network.

What is the difference between an infection of the cystic duct and of the gall-bladder? The same difference as between the cervix of any organ and the other portions of its cavity. The cervix of every organ has a rich lymphatic supply. The cervix of the stomach, the cervix of the uterus and of the urinary bladder, all have a rich lymphatic supply. The cervix of the gall-bladder, which is the cystic duct, has a rich lymphatic supply. Absorption takes place readily, and its occurrence is manifested by chills and fever, and you can have chills without having symptoms of jaundice associated with it. Furthermore, in infection of the gall-bladder the patient can have chills and fever without gall-stones. One of the most severe cases of chills and fever in a biliary infection I ever saw never had a colic, never had jaundice, never had anything except the classic cholangic temperature to lead to the belief that the infection was in the biliary tract. There was no pain, no tenderness—nothing. That man was about seventy-eight years of age. For eleven years he had had chills and fever. He traveled all over the world to get rid of his "ague," but it kept coming back and back periodically. Finally he went to California to spend the winter. He began to have the chills and fever shortly after he left New York, and in California the chills and fever continued. I saw him in consultation and he had this type of temperature—he would have a chill and a temperature following it up to 105.5° F. It would remain high for three or four hours and then drop to normal, remain normal for ten days or two weeks, then go up again. It was not a temperature curve, but a temperature angle. In my description of that peculiar type of temperature in Sajous' Medicine I termed it the "temperature angle of cholangic infections." When I examined that man on the morning of the second day of an attack I could hook my finger deep under the costal arch, and with his body flexed forward, when he endeavored to take a deep breath, he felt a distinct soreness. Then we made a diagnosis of gall-bladder infection. It was suggested that he go east to be operated on. He was a native of Connecticut, a hard-headed old Yankee who had built up a great fortune in his clean-cut, forceful way. At the second consultation we described his case to him, and he said, "You are satisfied that is what is wrong with me?" "Yes." "You think I should go home to be operated?"

"Yes." "Do you know what I think? You doctors think I am going to die and you are just going to get this old man off your hands because you don't want the responsibility of operating on him." He was right in that diagnosis even if we were not in ours. He said: "If it is agreeable to you I will go to the hospital across the street and have that operation tomorrow morning." Which he did. On opening the abdomen it was found that his gall-bladder was about as large as the two distal segments of my little finger, but there was no stone in it. The contraction was the sequence of a long infection, extending over eleven years. It was so small that we could not put in a drainage-tube, so we split it down lengthwise and sutured it around the tube with silk so that the gall-bladder would have to stay open and drain. The next morning he had a chill, with a temperature of 106° F. That was his last chill. That is many years ago, and he has had no return of his chills and fever. I merely cite that case to call your attention to the sparsity of symptoms that may be present, but that temperature is so typical, so classic, that you can make a diagnosis on the basis of the temperature alone. They never have jaundice, but they are always sensitive to my perpendicular percussion and subcostal hook-finger pressure *during the attack.*

Let me call your attention to the next class of cases in which you may have a temperature elevation, the cases in which the stone has passed into the common duct and is associated with infection. The patient has pain, nausea and vomiting, sensitiveness to percussion over the gall-bladder, of the perpendicular type which is so characteristic, chills and fever, and jaundice. That type of chills and fever with jaundice was early recognized in this country as malignant malarial jaundice. You can see why. The chills and fever were of the same type as those of malaria associated with jaundice, which were described in the text-books during the decad from 1870 to 1880. Trousseau mentioned this type of jaundice, associated with chills and fever, and he believed that the lesion was a lesion of the duct, but Dr. Quine, of this city, was the first man to put into the English literature four cases of stone in the common duct with the typical chills and fever, in which autopsies were made and an ulceration of the common duct found caused by a stone. This caused the chills and fever, so that in place of being a malignant malarial jaundice, it was an infection of the common duct, which has a rich lymphatic supply, thus clearing up the situation entirely. It differs entirely from the melanotic jaundice of malaria in its clinical course.

In this particular case the patient had these recurrent pains up to the nineteenth of March, 1913. He never had a jaundice. He had twenty years of pain, nausea and vomiting, local sensitiveness, but no elevation of temperature and no jaundice, all preceding the escape of the stone from the cystic duct into the common duct; then on the nineteenth of March of this year the stone, which was really a wedge-shaped mass made up of a number of small stones, passed from the cystic duct into the common duct and then he had his first manifestation of jaundice. He had an intermittent type of jaundice—not the type of jaundice that comes with a carcinoma. That went on until this morning. After the stone passed into the common duct he had the manifestations of infection—chills and fever. The chills and fever would disappear after a week or ten days, then come again and disappear again, and so on. We knew that this was an obstruction of the common duct and not a carcinoma. We laid down a law in our article in Sajous' Medicine which is always true, with the few exceptions to prove the truth of the law. What is it? A jaundice preceded by a colic is always a foreign body jaundice. Is that clear? It is a foreign body in the duct that causes the obstruction and the colic and the jaundice. A jaundice that is not preceded by a colic is never a gall-stone jaundice, but is caused by a lesion of the pancreas, by a tumor from without obstructing the bile by pressure, or an infection within causing such a swelling of the mucosa as to occlude the lumen of the duct. The colic is not due to the fact that bile is retained in the biliary passages and ducts: it is caused by a moving body in them. The colic is symptomatic of a foreign body being forced on through a tube whose lumen is smaller than the diameter of the foreign body. It does not matter whether the foreign body is in the ureter, the intestine, or the biliary tract, the result is exactly the same. The patient has a colic and the colic continues only while the foreign body is in transit, never after it is arrested for a considerable time.

Many of you remember my speaking of a patient who had a piece of plum pudding in the intestine for twenty years. I took it out of the intestine twenty years after he had eaten it. It was in that intestine all that time. During that time he had 15, 20, or 30 attacks of colic a year. Every time the mass moved on he had an attack of colic. It was so large that it could not be evacuated. It had formed numerous diverticula, and so long as the intestinal contents could pass by it the patient was all right, but when it moved into a narrower part of the bowel, he had a colic. I removed it from the ileum four inches above the ileocecal valve, the narrowest point in the small intestine. I want to impress this on you in connection with all these colics—*the colic does not continue when the body is not moving*. The clinical fact is that the colic is not present when the stone is not in transit. We had another case of the same kind, where the stone had passed out of the gall-bladder, ulcerating through its wall into the small intestine, and then she had the colic. She had repeated attacks of colic after that, extending over a period of months, until finally she became so ill that she had to have an operation. A gall-stone the size of a bantam's egg was removed from the ileum. She was not having the colics while the material was passing by the stone while it was in a diverticulum.

That is just what happens in the gall-bladder. You can see why this man is jaundiced. From the fact that the jaundice was preceded by a colic you know that it was primarily a gall-stone jaundice. On account of the long infection of thirty-five years we feared he might have developed a carcinoma in connection with it. He had a rather thickened pancreas, probably the result of infection, but no carcinoma. Notwithstanding the infection in the head of the pancreas, he has no indications of carcinoma. The man is now sixty-nine years of age. He was operated in the presence of jaundice with infection, which is one of the most dangerous conditions under which a gall-bladder operation can be performed. He had a stone as large as my thumb in his common duct. We still fear an infection that may occur and ascend to the liver and end his life.

You can see in following that case that you have the history of typhoid; the history of stone in the gall-bladder; the history of stone in the cystic duct; and the history of the stone finally reaching the common duct, with infection. We removed a great mass which was made up of a number of smaller stones.

Let us read the history of the next case and see what a picture that presents. These are both classic histories. If you get these two cases clear in your mind, you will have a fairly good picture of two varieties of lesions, namely, gall-stone in the common duct and gall-stone in the gall-bladder.

HISTORY

Patient, female, aged forty-six, enters hospital on account of attacks of sharp pain in the right hypochondriac region, followed by nausea and vomiting. In November, 1912, the patient had a chill, followed by pain in the wrist and forearm, with swelling of the wrists. She had no sore throat or cold preceding this trouble. The chill was followed by a temperature of 103° F. She had a dull pain in the right hypochondrium at that time, with tenderness on pressure. She became jaundiced after three or four days, and it continued during the next two weeks. The latter part of December, 1912, after auto-riding in the afternoon, she had a severe pain in the right hypochondrium, with nausea and vomiting a short time (about one hour) afterward. She also had a chill, followed by a temperature of 102°–103° F. She had chills every two or three days for the next three weeks, and a temperature elevation for four weeks. Patient had a number of attacks of sharp pain followed by nausea and vomiting during the next three or four weeks. She would have from one to four attacks a day, and become very icteric on the third or fourth day, with large red spots all over her body. She does not know what the color of the stools was at this time. She was tender to pressure over the right hypochondrium. During March, 1913, she had attacks of sharp, colicky pain in the right hypochondrium four or five times a week. The pain would persist for from four to five hours. The last attack occurred in the latter part of April, about two weeks ago. The pain was severe. The patient was nauseated, but did not vomit. She has been slightly jaundiced since the attack on January 2d. The jaundice has become a little worse in the following attacks. Patient had spinal meningitis fifteen years ago; was operated on for appendicitis five years ago. Had gravel in the urine two years ago.

Note.—A culture made from the gall-bladder contents aspirated at the time of operation showed staphylococci.

Dr. Murphy: When we examined this patient she was sensitive to perpendicular percussion over the gall-bladder. That is your cue to the diagnosis at once. Have the patient take a full breath to push the gall-bladder below the costal margin, and while she is in forced inspiration produce a perpendicular percussion according to our method. If the gall-bladder is diseased or the seat of disease, this percussion causes pain so severe that she will reprimand you for hurting her. That never occurs when the lesion is in the appendix. You can make this test at the bedside in the acute cases better than at any other time, because then the gall-bladder is distended. It is just like percussing over a distended urinary bladder.

She was not jaundiced so that her neighbors could notice it. She was subicteric—not jaundiced, as was the first patient. This subicteric condition is commonly present when there is an infection of the biliary passages which produces an edema of the common duct up to and including the opening into the duodenum. Just enough bile passes into the bowel so that there is not a complete obstruction, otherwise the patient would have an obstruction jaundice, as one sees in the cases of stone in the common duct with obstruction. What else did she have? She had an infection of the neck of the gall-bladder. Why? Because she had the typical chills and fever, showing that she was absorbing infectious products. And, finally, she had attacks of colic, which meant that she had a moving foreign body in her biliary passages. The fact that she was slightly jaundiced indicates that the stone was not large enough to produce a complete obstruction. She came into the hospital in the interim between attacks. We operated. What did we find? A band of adhesions extending across from the duodenum over the gall-bladder,—a non-compressible gall-bladder. That represented nature's effort to wall off the infection and keep it from extending to the neighboring peritoneum. What else did we find? A non-compressible gall-bladder. As soon as we relieved the adhesions, which extended down to the cervix of the gall-bladder, the gall-bladder was readily compressible. Before it was compressible we could not demonstrate stones in the gall-bladder. As soon as we relieved the tension, then we found quite a large number of stones. The pericholecystitis was the result of the infection in the gall-bladder passing through its wall. We removed the stones and drained the gall-bladder.

I cannot leave you this morning without telling you of a patient we had a few days ago. She came in for a diagnosis. She had pain of the recurrent type in the left side of the abdomen. I examined her and it seemed to me that the trouble was in the left kidney. I could not outline the left kidney definitely, and fist percussion over the left kidney did not give any particular information. She was only slightly sensitive. We told her that we would do a ureteral catheterization the following morning to determine whether she had a lesion of the left kidney. During the night of her arrival at the hospital she had a typical renal colic. When I struck the kidney with the fist percussion the following morning it gave her severe pain. She cried out. That told the story. She had an obstruction in the left ureter or in the pelvis of the left kidney. When I struck her with my fist over the kidney she had pain. I struck a distended renal pelvis: exactly the same proposition as in the case of the distended gall-bladder. That told us at once where the lesion was. We did not have to catheterize the ureters. Fist percussion is one of the most valuable signs of determining an obstruction to the renal pelvis or the ureter clear down to the bladder. The examination in the attack differs so from the examination in the interim, and I want to impress on you that you can do many things at the bedside without the microscope, without the instruments of precision, that will help you to make a definite diagnosis. Our diagnosis in this case was confirmed by examining the urine. It showed twenty or thirty white cells to the $\frac{1}{6}$ field, with some red cells, because she was in the acute obstruction stage. We will have a skiagram made of that patient to see if a stone is present. Then we will operate for the removal of the obstruction to the pelvis of the kidney.

I bring these cases before you because they are so plain and because they represent a classic symptomatology leading to classic pathologic entities. The doing of the operation is a matter of minor importance which you can soon learn on the cadaver and the animal. The diagnosis is of major importance and can be acquired only at the bed-side and with the patient present.

STUDENTS' CLINIC AT OPENING OF SESSION THIS YEAR

Illustrating Dr. Murphy's Method of Student Instruction. With Preliminary Remarks

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Dr. Murphy: In our plan of teaching clinical surgery in this clinic the purpose is to get the student *to think*, and to think not only when he is in his seat, but also when he is on his feet, which is a much more difficult proposition. When I press him for words and for answers, remember it is always impersonal; we are not endeavoring to put him in the position of one who does not know, but to get him to think and to get the other members of the class to think with him along the line he is trying to think on. We do not expect a man in the arena to do the best work he is capable of doing. We know he will not answer as well at that time as he is capable of answering on the benches. We ask him questions for the purpose of fixing the attention of the others who are in the seats.

Remember, the best way to learn is as the child does, *learn by asking questions*. Learn to ask and answer questions and do not be satisfied until you can do so. Remember that, as students, you have the same right to ask questions when you do not understand that we have to ask you questions. When you go home at night, if you have the time to look up the subjects that were discussed here, it would be very advantageous to you. If you will read up the subject in some short text-book, not the larger systems of surgery, but one of the short text-books, you will get some important information in relation to the pathology, symptomatology, diagnosis, and treatment. While a surgical clinic is ostensibly an operative clinic, this clinic is not an operative clinic. You should go to a surgical clinic to learn pathology, applied anatomy, applied physiology, and, above all, applied diagnosis, rather than how to operate. The mere operative procedure is incidental. You will see some operations in this clinic, but not many, because that is not the purpose of this clinic. You can learn to do operations after you have graduated and have time on your hands. You cannot learn diagnosis anywhere as well as in the clinic, where you have the patient before you. The purpose of reading up at night on that particular case is because you have seen the case. The knowledge you get about that case is *your* knowledge, and not book knowledge. It is a *personal* observation knowledge, not reading knowledge.

In medical schools we make a practice of confining the student's attention during the first two years to the laboratory and scientific branches without coming in contact with the patient. I think there will be a change in that system in the near future. We are going to apply this scientific knowledge to the patient's case from the very beginning. It is the application to the patient of the essentials that you learn in the laboratories of anatomy and physiology that makes the study of these subjects of value to you. Possibly you know anatomy and physiology as abstract sciences, but you are taught these subjects for the purpose of applying them to clinical cases. It is not probable that you are going to devote your life merely to learning these subjects for the pleasure of an abstract knowledge —you are going to apply them practically for clinical purposes. The stimulation you receive from the study of these subjects in the early days of your college career aids you in your later work at the bedside.

If you were to ask me to recommend the best book on surgery, I would say that the best surgery is a good physiology, the best you can buy. Next to that is the best pathology. These two of necessity demand a third, a text-book on anatomy.

Following out that line of thought I wish you to keep in mind all the time that surgery is drifting continually from the mechanical to applied physiology. For a time it drifted to applied pathology, then from pathology to applied physiology, with pathology and anatomy as secondary requirements. When I was a student the man who went into surgery came in through the route of the professorship of anatomy. The man who was the senior assistant in anatomy became the professor of anatomy and finally professor of surgery. We have not yet heard of the professor of physiology becoming a surgeon. The professor of physiology becomes the internist, the diagnostician, but the professor of physiology will sooner or later come into surgery as his own—not as high a calling as that of internal medicine, and never as high as diagnosis.

There is nothing so important or practical or intellectual in medicine or surgery as the interpretation of symptoms and physical signs into pathologic entities. It is around this that most of your practical work will revolve. It is around this that most of your reading will revolve. You cannot attain that unless you have your physiology, your biochemistry, your pathology, and your applied anatomy well grounded.

In the study of surgical diseases ask yourself questions. Ask your neighbor questions. There is nothing about surgery that is obscure or hidden. Simplify everything. Bring every pathologic process that you have to deal with, that is hidden in the body, metaphorically to the surface and say to yourself, "If that were spread out here on the table, how would it look? how would infective products destroy the neighboring tissues? how would they metastasize to other portions of the body? what would I do to limit the metastases into the neighboring tissues or the destruction of tissue? what would I do to relieve it?" Do not look at it as something hidden—bring it to the surface. Make a mental picture of it as if it were spread out on your hand. By doing that you will have an entirely different conception of disease. Many things are done in medicine and surgery in the case of disease in an obscure location that would not be done if these same lesions were brought to the surface or were similar lesions on the surface. They would be treated in an entirely different and more intelligent way. This is for the purpose of simplification. For a time we leaned to complex operations, complex technic, complex instruments, and, above all, we leaned to mechanically made and machine-made diagnoses. Now we are making diagnoses with cortical cells. Remember the patient is the unit around which all your life must revolve—scientific, moral, social, and otherwise. Your obligation always comes back to the individual patient. That is the purpose for which you study medicine, to *benefit the patient.*

For a long time—nearly twenty years—it was considered the highest intellectual attainment for the medical man or the surgeon to be a therapeutic nihilist. That feeling has vanished now or is vanishing rapidly. We are not satisfied with simply making a diagnosis. Here there is intelligent therapy and here the patient must have thoughtful consideration, more than the mere determination of what is the matter with him. Ascertaining what is the matter with him is only the stepping-stone to doing something of definite value for his improvement. I wish to impress that on you probably more than any other one thing that I mention. In order that you may render the best service to the patient, you must not only know your pathology, physiology, and anatomy, but you must know the clinical course of the disease so that you will be able to estimate where you can check its advancement, change its course for the better, or where you can remove that lesion. Not only where, but you must know when. *You must act in time.*

If you were to ask me what is the cause of the greatest mortality in surgery, I would say that it is "procrastination" —delay. There was a time, and that just about two decads ago, when it was quite the proper thing for the surgeon to procrastinate and by this indicate his conservativeness. That was a misnomer. He was not a conservative because he procrastinated: he was a conservative because he was a scientific coward. He did not have the confidence in his ability, either as a diagnostician or as a surgeon, to do the things that should be done early. He did not have the courage to "pick pathologic processes when they were green." He waited until they ripened, until there had been a considerable degree of destruction of tissue. If you are to be a surgeon who is of the greatest possible service to the people, you must eradicate pathologic processes or check them when they are in the initial stage, before the patient's resistance has been lowered, before metastasis has taken place, and *before* *the patient's life or limb has been hazarded by the process.* If you, by your procrastination, hazard that patient's life by waiting, you are just as guilty as if you hazarded it by an erratic action. Remember, the sins of omission are very considerable ones in the practice of medicine and surgery. They are going to be recognized not by the profession alone as reprehensible, but they are going to be recognized by the State and the people as such. The people, the courts, and particularly the conscience of surgery will demand the recognition of disease in its earliest stages—the interpretation of symptoms and signs at their very earliest stage into their different pathologic entities. That is the principal aim of your work in the clinic, to endeavor to interpret the history and clinical course into pathologic entities.

[Here Dr. Murphy asked five students to come down into the arena to take charge of the first patient.]

METASTATIC CARCINOMA OF FEMUR

History

Patient, a female, aged forty-eight, a widow, enters the hospital on account of pain in left hip, inability to walk, and marked weakness.

Previous history negative, with the exception of a radical operation for cancer of the right breast in October, 1907. Husband died two years ago of pneumonia. Married eighteen years, no children, no miscarriages. Menses began when sixteen years of age. Had normal menopause at forty-three. No history of tuberculosis or carcinoma in family.

Present trouble began the second week in July, 1912, when patient, after being exposed to dampness for about three months, was taken with sharp, stinging pain in the left hip, which radiated down the left limb posteriorly as far as the knee. Her physician told her she had sciatic rheumatism. She kept on working up to August 27th, when pain became very severe. She went to Benton Harbor and took the baths for two weeks, but felt no better. She came home and then was obliged to use a cane to get about. She went to a hospital on October 24, 1912, and left November 21, being treated for sciatica. She was very much improved. Had no pain with the exception of a slight sting at times, but she had to walk with a crutch because of weakness of her leg. [This should have been in itself an indication that there was a more serious lesion present than sciatica.—Ed.] The first Sunday in March, when going to bed, her crutch caught on the door-sill, she fell forward and was thrown against the dresser, striking her left hip. She kept herself from falling to the floor by grabbing the knob of the door. She immediately had great pain at the site of the injury [which was the position of her former pain in the upper part of the thigh. —Ed.]. She dragged herself to a chair and remained there all night until put to bed by her family the next morning, and she has been in bed ever since. This pain lasted four or five weeks. She was taken to the County Hospital April 7th and left there August 14, 1913.

On examination there was found an apparent shortening of left limb, 1½ inches; a hard, bony tumor mass, the size of a fist, on the upper end of the femur; slight pain on rotating limb outward.

Does not know about the loss of weight or anything else in connection with the development of tumor on the hip.

Dr. Murphy: What is the matter with this patient? [To the patient]: How old are you?

Patient: Forty-nine.

Student: How long have you been sick?

Patient: Nearly a year. I was treated for sciatic rheumatism last November.

Student: Did you have pain before last November?

Patient: I had pain since about August.

Student: That is the first time you had that pain?

Patient: Yes.

Student: Has the pain persisted ever since?

Patient: Off and on.

Student: What time does it come on particularly?

Dr. Murphy: Start with that patient at the beginning of her trouble and proceed with her up to the present time. That is the way to make a diagnosis. Take the symptoms and signs in the order in which they occur. Travel with her, first ascertaining the beginning of the trouble.

[To patient.] In August you began to have pain?

PATIENT: Yes.

DR. MURPHY: Travel with her from the first day the trouble appeared in July, 1912, until this third day of October, 1913.

STUDENT: How did the pain first begin?

DR. MURPHY: She worked in a basement and felt it was from the dampness. Why do you remember the twenty-seventh of August?

PATIENT: That was the day I stopped working.

DR. MURPHY: Why did you stop then?

PATIENT: I was going to Detroit.

DR. MURPHY: Did you leave because you were ill?

PATIENT: I had pain in my hip.

DR. MURPHY: Pain in your hip on the twenty-seventh of August. Was that pain sufficiently severe to compel you to leave work? When did you have the pain—in the daytime or at night? Did you injure your leg by falling?

PATIENT: No.

DR. MURPHY: When you got to Detroit, could you walk?

PATIENT: I walked to the depot here. When I tried to go home, on arriving in Detroit, I could not walk.

DR. MURPHY: Did you have an accident after you came back to Chicago?

PATIENT: My crutch stuck in the door, I fell against the bureau and struck my hip.

DR. MURPHY: Did you fall down?

PATIENT: No.

DR. MURPHY: Could you put weight on your leg after that day?

PATIENT: No, I was in bed five weeks.

DR. MURPHY: *Her leg struck against the dresser, but she did not fall down.* She was in bed five weeks after that. What happened at the end of five weeks? While she was still in bed, was she able to lift her foot off the bed? Go on, doctor.

STUDENT: Could you lift the left foot up?

PATIENT: No, I could not. My leg was lame.

DR. MURPHY: How was the sciatica after that fall? Did you have more pain?

PATIENT: No, doctor.

DR. MURPHY: How was the pain in your leg while you were in bed?

PATIENT: I do not recall it.

DR. MURPHY: But you could not lift the limb off the bed. Could you lift it over the side of the bed?

PATIENT: All my limb hurt me.

STUDENT: What happened at the end of the fifth week?

PATIENT: I could get up and walk.

DR. MURPHY: Were you able to do this gradually? Did the hip pain after you were able to walk?

PATIENT: I had no pain.

DR. MURPHY: Could you put weight on the limb?

PATIENT: No, sir.

DR. MURPHY: First you walked with a crutch. Did you have two?

PATIENT: One.

DR. MURPHY: One, so she had to put some weight on the limb necessarily. On what date did you fall? Was it in November?

PATIENT: No, in March, 1913.

DR. MURPHY: Did you have pain all over after you fell in March? Have you walked on the limb since?

PATIENT: Yes.

DR. MURPHY: Without a crutch?

PATIENT: No, I had to have crutches.

DR. MURPHY: What is the matter with her?

STUDENT: Were you able to walk alone?

PATIENT: No.

STUDENT: Were you in the same condition when you were in bed?

PATIENT: I had no pain.

DR. MURPHY: The pain has not entirely disappeared, but you never had as much pain after the fall as you had preceding the fall?

Patient: No.

Dr. Murphy: Correct. What is the matter with her?

Student: Were you able to move your injured limb at all? Has it given you pain all the time?

Patient: Yes, sir.

Dr. Murphy: What do you notice when you look at the hip? What about the conformation of that limb?

Student: It is curved outward.

Dr. Murphy: Yes, there is a deformity in the alinement. It has a decided curve, is not that right? What is the matter with her?

Student: It is either a dislocation of the head of the femur or a fracture of the neck of the femur.

Dr. Murphy: Show me what dislocation of the femur would produce the deformity that you have here. Just demonstrate it to the class. What luxation of the femur would produce that deformity?

[Demonstration.]

Student: It would go into the ligament of Bigelow. It would have to pass upward.

Dr. Murphy: If the head passes forward, where would the shaft be? It would necessarily be back of the normal alinement of the femur. How will you get the shaft up in front to make that elevation so that the head would be behind? If there is a luxation here, how are you going to represent it? Here is a skeleton.

Student: Probably it is in a posterior position.

Dr. Murphy: Put it in position. I am ready to accept it. If it is luxated back in that position, this shaft would come too far in front of the line of the trochanter. It would go clear back, then what would happen to it? Go farther down—what would happen to it? Put it in here, what would happen to it?

Student: Still the trochanter will be behind.

Dr. Murphy: Yes, the trochanter would be behind, and on the patient it appears to be in the proper relationship to the anterior superior iliac spine. The upper portion of the trochanter is a little higher up than normal. It is ½ to ¾ inch higher up.

STUDENT: She has a fracture there.

DR. MURPHY: All right, I am open-minded. I am waiting to be persuaded what it is. I will believe you. What about it?

STUDENT: It looks like a fracture of the upper end of the femur in about this position [indicating upper end of shaft just below lesser trochanter].

DR. MURPHY: If it were a fracture of the neck of the femur, where would the trochanter be?

STUDENT: Up in the normal position.

DR. MURPHY: No, higher up. Why do you think the fracture is in the upper end of the shaft of the femur? There was a destruction of the normal line or the contour of the shaft of the bone, is not that true?

STUDENT: Yes, sir.

DR. MURPHY: If the shaft is pushed up, you have the head in an anterior relationship. She is able to move the leg around pretty well, but there is a false point of motion in the shaft as well. How long since she had the accident?

STUDENT: In March, 1913.

DR. MURPHY: And she still has a false point of motion here. What is the matter with her?

STUDENT: She has a fibrous union.

DR. MURPHY: She would not have that much motion with a fibrous union.

STUDENT: I mean a non-united fracture.

DR. MURPHY: All right. Before you have a non-united fracture you must first have the fracture. Is not that right? Did she have a fracture?

STUDENT: Is this the point where you fell against the bureau?

PATIENT: My hip struck against it.

DR. MURPHY: What is the matter with her?

STUDENT: A fracture of the upper end of the femur.

DR. MURPHY: Why did she have crutches *before* she had the injury? It is hardly the way of things to commence to use crutches before you get the fracture. That is why we are beginning to study disease in the order of procedure—the symptoms. That is what makes a diagnosis.

STUDENT: The use of the crutches was due to the severe sciatica she had.

DR. MURPHY: That is right. What do you mean by that?

STUDENT: An inflammation of the sciatic nerve.

DR. MURPHY: What makes you think she had that? Because she told you so? Did you have pain in the back of the leg?

PATIENT: All down in the hip.

DR. MURPHY: From the hip down. Did the pain go as far down as the toes or heel before you had the fall?

PATIENT: No, it was just in the hip.

DR. MURPHY: But you seemed to be more tender in the back of the leg than in front. Where was the tenderness to pressure before you had the injury?

PATIENT: Down along the back of the leg. That was the sciatica.

DR. MURPHY: That is what it was called. That is what the doctor called it. What is sciatica?

STUDENT: Inflammation of the sciatic nerve.

DR. MURPHY: No, not at all. "Sciatica" is just a pain in the neighborhood or in the course of the sciatic nerve. That is what sciatica is, as it is generally understood. What is the pathology of it?

STUDENT: Inflammation of the sheath.

DR. MURPHY: Not at all. The pathology of "sciatica" is no definite pathologic entity at all. It is merely a pain manifestation due to many conditions. It is one of the symptoms of many lesions in the lower extremity, anywhere from the second lumbar vertebra down to the toe. When you make a diagnosis of "sciatica," you make a very wise statement to the patient, but when you ask yourself, what is it? what is it? what is it? you have to admit that you did not find out what it was. To call it sciatica, is just like saying "neurasthenia" or "malaria." It does not mean anything, except a word to cover up the absence of real knowledge. Now do not be misguided by it. Do not use it. Find out what lesion is producing the pain and do not merely call it neuritis or neuralgia. If it is neuritis, what causes the neuritis? What is the lesion? Has the patient an osteitis, a periostitis, or a traumatic synovitis, or an intestinal obstruction that is pressing on or interfering with the function of that nerve, or has the patient a lesion of some more serious character in the joint supplied by that nerve? Remember sciatica is a symptom only. If you use the word as a symptom, the same as you use ileus, explaining a syndrome of symptoms, then you will soon commence to find out the definite pathology from the story of the symptoms. What is the matter with her?

STUDENT: Has a blood count been made?

DR. MURPHY: Yes, what is it?

INTERN: Seven thousand leukocytes; erythrocytes, 2,400,000.

DR. MURPHY: Was a differential white count made?

INTERN: No.

STUDENT: Any temperature elevation?

DR. MURPHY: No.

STUDENT: Was the urine examined?

DR. MURPHY: Yes, negative.

STUDENT: Were the lungs examined for a tuberculosis?

DR. MURPHY: I think not. She has no cough, no temperature elevation, no expectoration. You are a long way from the trouble. Go back again to the day of, but preceding, the accident. She had so much pain in her limb that she could not put weight on it. She used a crutch—one crutch—in order to walk. Every time she put weight on the leg she got the sciatic pain, as she uses the expression. Did she have a synovitis or some lesion of the thigh? Then we come to the accident. She stumbled but did not fall. Following that she was unable to lift the leg off the bed, unable to walk for five weeks, and from that time on she had the deformity.

STUDENT: Did you feel a grating of the bones when you fell?

DR. MURPHY: She does not think so.

STUDENT: Did you feel a crepitation as though you had two surfaces rubbing together?

DR. MURPHY: She does not recall it. At the end of the five weeks the severe pain disappeared. After what? After the fall.

Note that she never had a severe pain, except at the moment of the accident. Finally she got up, but could not put weight on the limb, and now she comes in here with a deformity. What is the matter with her? Did she have a sufficiently severe injury to break a normal femur?

Student: Yes, sir.

Dr. Murphy: I do not think so. She was walking with her crutch, fell, and struck her hip against a bureau and the femur broke. A femur does not break that way if it is a normal femur. If it is not a normal femur, had she a lesion in the femur that predisposed it to fracture? What was the lesion that caused the sciatic pain, and why was the pain less severe after the fracture than it was before she fell? Was a growth present somewhere in that femur?

That is a typical history—a classic history—of a pathologic fracture, for the injury was not sufficiently severe to produce a fracture in a normal bone. When you look at the skiagram, you will note that the trochanter is higher up than normal, as we estimated it from the examination. There is a good apposition of all of that portion of the bone. The shaft of the femur is fractured. There is an enlargement of the upper end of the bone. Reading a skiagram is something you have to be trained in just as much as in interpreting signs and symptoms. What pathologic lesion caused this fracture?

Student: It might be a benign or a malignant tumor of the bone.

Dr. Murphy: Very well, tell us about that.

Student: A malignant tumor is generally of the sarcomatous type, shows rapid growth and early metastases. A benign tumor is generally of the osteoma type, slow growing, and does not produce metastases.

Dr. Murphy: All right, what is the matter with her?

Student: It is a bony growth producing pressure on the nerve.

Dr. Murphy: What nerve?

Student: The sciatic nerve.

Dr. Murphy: I do not think so at all. I do not think she ever had any pressure on the sciatic nerve.

Student: Have you lost any weight?

Patient: I do not know.

Dr. Murphy: What is the matter with her?

Student: Were there evidences of metastases anywhere else, doctor?

Dr. Murphy: No, sir. What is the matter with her?

Student: I think it is a sarcomatous growth.

Dr. Murphy: Just what makes you think so?

Student: The extreme pain.

Dr. Murphy: Which is not very common with sarcoma.

Student: The rapid destruction of the bone itself.

Dr. Murphy: I do not know how rapid that was. She had pain for a number of months, then she finally had a fracture. A sarcoma of the bone usually follows an injury, not precedes it.

Student: Yes, sir.

Dr. Murphy: There is no history of injury of the femur preceding the injury that caused the fracture. *A sarcoma never originates in a bone that is fractured. You understand an injury of mild degree causes a sarcoma and an injury of sufficient severity to produce a fracture never causes a sarcoma.* Put that down in your book. If the patient is struck with a baseball or strikes his shin on the corner of a chair, he may get a sarcoma. If he has an injury severe enough to produce a fracture, it never results in sarcoma; therefore, if she had a fracture originally without a disease of the bone and has a tumor now, it should not be a sarcoma. What malignant diseases are there of bone other than sarcoma?

Student: I do not know.

Dr. Murphy: How many types of malignant disease are there?

Student: The primary division of malignant diseases is into two types—the epithelial and the connective-tissue types.

Dr. Murphy: That is right, cancer and what?

Student: Sarcoma and cancer.

Dr. Murphy: We place endothelioma and hypernephroma

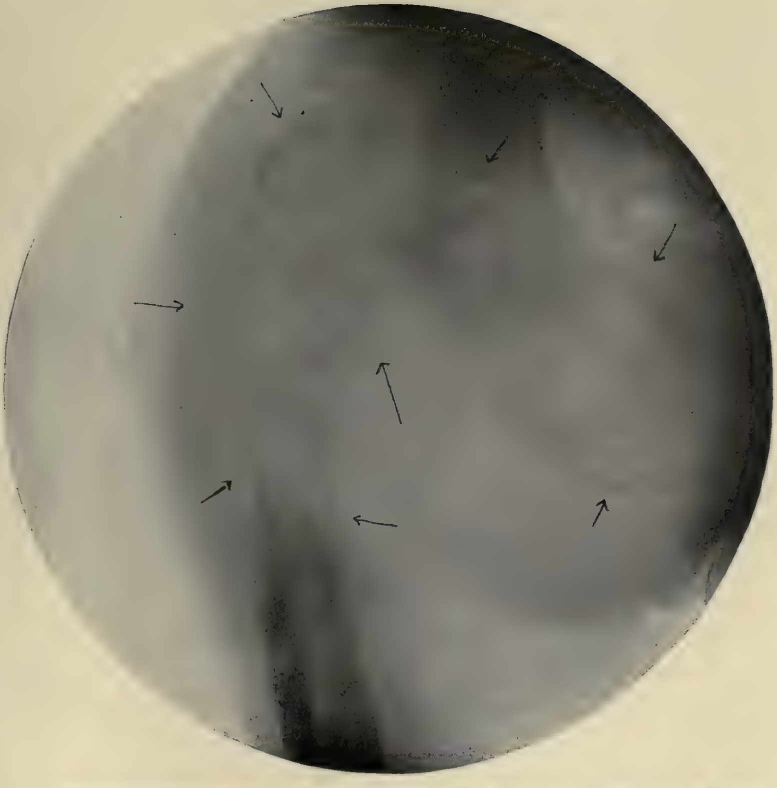

Fig. 256.—Metastatic carcinoma of upper end of femur, extending from below the lesser trochanter up to and including the acetabulum and pelvis, with a bowing outward of the femur.

under the connective-tissue type. We keep cancer of all varieties under the epithelial type. All right, what is the matter with the patient?

Student: Has she any situations leading up to this? There might be a carcinoma of the uterus or the stomach.

Dr. Murphy: No. She has neither. What is the matter with her? Does not the skiagram show that in addition to this fracture there is also a neoplasm there?

Student: Yes.

Dr. Murphy: All right, if she has a neoplasm she has one of two types of tumors if it is malignant. Is not that true?

Student: Yes.

Dr. Murphy: If there were other lesions that caused the enlargement and softening of the bone other than a malignancy what are they?

Student: Tuberculosis.

Dr. Murphy: Yes, tuberculosis is one, actinomycosis is another. Then there are the parasitic lesions, such as echinococcus, not at all uncommon causes of softening of the bone. When you look at this skiagram, you will see that it is not the picture of an echinococcus cyst. To show you the other lesions, I had this list of bone tumors put on the board. [See October Clinics.] She had no typhoid, no specific infection, no actinomycosis, no staphylococcus infection. The next question is, has she an osteitis fibrosa cysticus? The skiagram is not typical of that condition. Next on the list is chondroma. Then you have the thyroid tumors of the bone. What do you understand by that?

Student: A metastasis of the normal thyroid tissue into bone.

Dr. Murphy: Yes; where do they take place most commonly?

Student: In the long bones.

Dr. Murphy: In the long bones most commonly when malignant. If you have a carcinoma or a sarcoma of the thyroid, then it metastasizes much more commonly into the long bones, into the shaft of long bones, particularly in the metaphyses, which means the ends of the long bones, but the non-malignant variety, that is, the histologically typical thyroid neoplasm, metastasizes most commonly in the bodies of the vertebræ and on the surface of the calvarium. The malignant variety is an entirely different proposition. The malignant variety, if it is a sarcoma, metastasizes to the shaft of the long bones; if it is a carcinoma, it also goes to the shaft of the long bones. Why? What is the physiologic relationship—coming back to the application of physiology—what is the physiologic relationship between the thyroid and the long bones? What happens if you take out all the thyroid tissue from a litter of greyhounds? They will all develop into dachshunds. You can control the growth of their long bones by removing the thyroid, and you can start these dogs to grow at any time by feeding thyroid. So you see there is a direct physiologic relationship between the thyroid gland and the shaft of the long bones. There is also a direct pathologic relationship between the thyroid and the shaft of long bones. Has she a lesion of the thyroid of marked character? No. After you have gone over this wide range you come back to this individual patient. What is the matter with her? Next gentleman, will you kindly take up the subject and tell the gentlemen what is the matter with this patient?

STUDENT: I do not know.

DR. MURPHY: Next.

STUDENT: I do not know.

DR. MURPHY: Is that what you would say if she came to your office the first day you were practising and you were out of funds? I think not. You would endeavor to find out.

STUDENT: I think she has a destructive process of the head of the femur. The fall was due more to the fracture than the fracture to the fall.

DR. MURPHY: All right; what is the matter with her? We rather came to that conclusion—that she has a pathologic fracture. What is the destructive process? If it is a pathologic fracture, what is the indication for treatment?

STUDENT: The indication is to go in and remove that lesion in that leg.

DR. MURPHY: All right, I will do that. Sister, have you the bone instruments? Sister has not the bone instruments—that is a sign that there is no indication for that line of treatment. What is the matter with her?

STUDENT: I think the diagnosis lies between a tuberculosis and some form of malignant tumor.

DR. MURPHY: All right, her response to tuberculin is negative.

STUDENT: Then it lies with a malignant disease.

DR. MURPHY: What is the matter with her? It may be an echinococcus cyst and it may be a malignant tumor. Which is it?

STUDENT: I do not believe it is possible to say it is a sarcoma or carcinoma without operating.

DR. MURPHY: You do not? You will be sorry very shortly for making that statement. A sarcoma is a primary tumor of the bone. A carcinoma is what?

STUDENT: Usually secondary.

DR. MURPHY: Not usually, no. *Always.* That is right. That is positive—one is primary, the other is always secondary. Now, then, if she has a carcinoma of that femur she must have a primary carcinoma in some other portion of the body. Has she?

STUDENT: I do not think the history showed it.

DR. MURPHY: But there are a lot of things not brought out in the history. You determine it.

STUDENT: Has she lost weight?

DR. MURPHY: No, she said she lost weight first but not since.

STUDENT: She said she had pain.

DR. MURPHY: At first she had severe pain, but that has disappeared since the fall.

STUDENT: Have you lost in weight lately?

DR. MURPHY: Yes, she told you that. She has been losing, otherwise she is normal.

STUDENT: That might speak for a carcinoma.

DR. MURPHY: Yes, many times.

STUDENT: She is the proper age for carcinoma.

DR. MURPHY: Yes, that is right. There are some veins in the neighborhood. That is merely suggesting things: you are no nearer the point. Get to the point! Start right. What are the organs that suffer most commonly from carcinoma in the female?

Student: The uterus and the stomach.

Dr. Murphy: Did you forget this woman had breasts? She may have had a carcinoma of the breast. The doctor asked me if she had a carcinoma of the uterus or stomach, but forgot to ask me if she ever had a lesion of the breast.

[Examining patient.]

I find that one breast perfectly normal. The other one is not there. What is the matter with her?

Student: That looks as though that breast was removed for a carcinomatous growth primarily.

Dr. Murphy: It does not look like that to me from any facts we have received. I would not make a diagnosis on that.

Student: When was that breast taken out?

Patient: Five years ago.

Dr. Murphy: The first question you ask yourself is—is there a return *in loco*. There is no return whatever *in loco*. There is no return in the axilla. The breast was amputated five years ago. How long did you have trouble in your breast before it was amputated?

Patient: About a year.

Dr. Murphy: Was it a lump?

Patient: A little lump moving around. Then I had pain at night and had to get up and walk.

Dr. Murphy: Did it ever break or ulcerate on the surface?

Patient: No. It was just a little hard.

Dr. Murphy: It was operated five years ago. How long after that was it before she had the first symptom of the sciatic pain, the most common expression used in connection with a metastatic carcinoma of the lumbar zone of the spine and of the upper end of the femur, and which often continues for weeks and months, even for years, before the true pathology of the lesion becomes known. How long did you have the pain before you injured the hip—about a year?

Patient: Just about a year.

Dr. Murphy: Now, then, there is the story. A diagnosis of carcinoma. That patient began to have that continuous pain at night. One of the first things that should be thought of in her case was, was there a metastatic lesion? A metastasis from the breast into the upper end of the femur is the most common osteal metastasis we have. At one time in this hospital we had five patients with metastatic carcinoma in the upper end of the femur, between the trochanter and three or four inches below it. In two of these cases the patients gave the history of trauma as the first manifestation. In two the patients had no knowledge whatever that they had a lesion in the breast. They did not suspect the breast at all. One, a woman of about forty, was playing tennis and strained her hip. From that day the pain became worse. She had an extension put on for the purpose of stretching the nerve. It was called a sciatica. Why? Because the etiology of the sciatic pain was not discovered; because the sciatica was interpreted as being a disease in place of a symptom. When we came to look for the cause of the pain in the hip, we found that she had a carcinoma of both breasts. The other patient, a sister of one of the members of our training-school, had a congenital aplasic breast—a failure of development of the left breast. She came into the hospital after she had been suffering from "sciatic" pain in her hip for four months. The pain had become intolerable. In looking for a cause of the pain, the aplasic breast was examined and a diagnosis of carcinoma was made. There was a metastatic carcinoma in the femur and the sciatic pain was due to that.

I merely bring those things out to show you that the sciatica is a symptom and not a disease, and to accentuate the fact that a fracture in a normal leg must have a sufficient element of trauma in it. This woman did not have a trauma sufficient to break a normal femur. She had only a trauma sufficient to break a pathologic femur. Then she had in addition, what? As soon as the bone fractured, the relief of tension on the neoplastic cellular elements inside of the bone relieved the pain, which is characteristic of pathologic fractures. Then immediately following the fracture the pain was less severe than it had been for months—all of which helps one to make the diagnosis that it was a neoplasm and a pathologic fracture. Then to find out the primary location of the tumor. The primary location was in the breast. About four and one-half years elapsed from the time she had the neoplasm in the breast before she showed the first symptoms of metastasis. That is rather a long time, but not at all too long a time for so thin a patient. Remember, cancer in thin people metastasizes slower and grows slower both in the original primary location and in the metastases than it does in fat patients. It is this type of patient that gives the larger percentage of cures, because the connective tissue that is formed around the original misplaced epithelial cells offers a resistance to the tumor, therefore it grows slower in the thin than in the fat. Remember the prognosis with a carcinoma is much more favorable in a patient of this size than in a patient weighing 160 or 180 pounds. There is no operation that should be performed on this patient. She has in all probability either metastatic lesions in her mediastinum, liver, spleen, kidney, or other organs which cannot be eradicated even if the one in the femur could be.

Let us take up the next case. The intern will read the history.

TRAUMATIC SARCOMA OF FEMUR

History

Patient, a male, aged thirty-nine, married, entered hospital on July 16, 1913, because of severe, continuous pain in left thigh, which was greatly exaggerated on walking and which was most severe in the upper third of the thigh.

Present trouble dates back to February 1, 1913, when patient was walking between two freight-cars and slipped on some ice, wrenching left thigh in trying to prevent himself from falling under the moving train. He did not fall to the ground and did not strike leg against anything. Immediately following this he developed a sharp pain on inner side of left thigh, which soon spread over the whole thigh and which was exaggerated on walking. He could walk on leg immediately after injury, but had to limp on account of the severe pain caused by putting weight on his leg. He also used a cane. Had no chills following and does

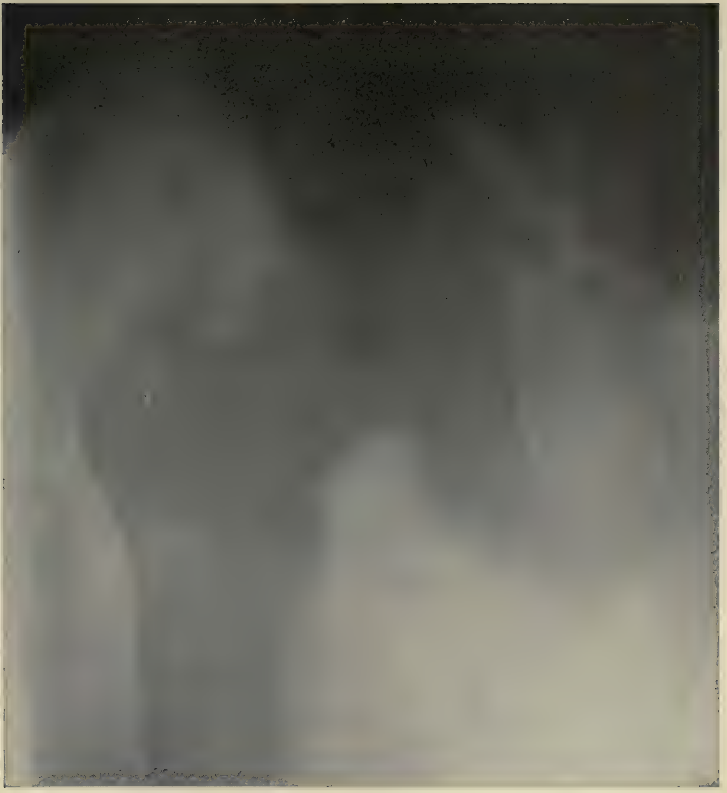

Fig. 257.—Traumatic sarcoma of femur. This and the succeeding three skiagrams should be compared, as they represent a progressing series of the same case. This skiagram was made soon after the injury. There is no evidence of tumor or disease of the bone.

Fig. 258.—Traumatic sarcoma of femur. This skiagram was made about four months after the injury. The dark shadow in the inner part of the shaft just below the lesser trochanter is the first evidence of the sarcoma.

not know about the temperature. No x-ray was taken. Three days later he consulted a doctor, who said he had muscular rheumatism and prescribed internal treatment and hot baths. Patient kept up this treatment and continued work until June 23, 1913, when pain became so severe that he had to quit work until July 1, 1913, but he was able to work only two days. He then went to a masseur and took baths until July 12, 1913. On July 16, 1913, he entered this hospital in another service. He had no temperature when brought in, and did not have any chills up to this time, but the pain still continued. One week following an x-ray picture was made, which showed a dark shadow in upper third of left femur. A few days following this some more x-rays were taken, which showed the same shadow, and a diagnosis of osteomyelitis was made. It was decided to operate on him on August 13, 1913. On August 12 the white count was 16,000; had no temperature or chills. On the night of August 12, 1913, while turning over in bed, the patient developed a severe pain in the upper third of left thigh, which caused him to flex the thigh on the abdomen. He could not straighten the leg right then because of the extreme pain. He did not feel any breaking or crepitation. The following night the leg was straightened under anesthesia. Immediately following this the leg began to swell and he began to have temperature, ranging between 99° and 101° F. He had no chills or sweats. No x-ray was taken. On Friday, August 15, an incision was made on the outer side of the thigh, and a large blood-clot was found and also a fracture in the upper third of the femur. There was severe bleeding, and the wound was packed tightly with gauze. This packing was removed piece by piece within the next four to six days, and the wound allowed to close by primary healing. After operation the temperature ranged between normal and 102° F. No chills. Extension was put on leg immediately after the operation and it has been kept on since. At present has very little pain and temperature is normal. The wound is not entirely healed.

Previous History.—Denies venereal infection. No lues. No previous sickness. No family history of tuberculosis or malignancy.

Dr. Murphy: Next gentleman, make a diagnosis of this case.

Student: How old are you?

Patient: Thirty-nine.

Student: How long have you been sick?

Dr. Murphy: Listen carefully to this story, because it is the story that is so important in making a diagnosis. [To patient.] You are a railroad man and you were running along the side of the train and slipped. What happened?

Patient: I slipped toward the train, and to avoid falling under the train I strained every muscle to save myself, and I wrenched myself terribly.

Dr. Murphy: Wrenched yourself where?

Patient: In the left thigh—high up in the left thigh.

Student: Did you have a good deal of pain at the time of the accident?

Patient: Yes, considerable.

Dr. Murphy: How long did the pain continue at the time of the accident? Were you able to get up and walk after the accident?

Patient: Yes.

Dr. Murphy: Did you limp the first day?

Patient: Yes.

Dr. Murphy: Did you limp the second day?

Patient: Yes.

Dr. Murphy: Did you limp two weeks after?

Patient: I do not remember taking a step since without limping.

Dr. Murphy: Was the pain two weeks after the accident as severe as it was two days after? How severe was the pain two months after?

Patient: It was steadily getting worse.

Dr. Murphy: What is the matter with him?

Student: When you fell did you feel that you had broken bones?

PATIENT: It did not feel like that. It felt as though the muscles were injured.

STUDENT: Were you able to walk at all?

PATIENT: Yes.

DR. MURPHY: What is the matter with him? What would you think from the history? You cannot inspect the leg because it has a dressing on it. It would not add very much light if you did. Did he have a chill following the accident? Did he have to go to bed?

STUDENT: Did you have a chill?

PATIENT: No.

DR. MURPHY: Did you have a rise in temperature?

PATIENT: No.

DR. MURPHY: Why did I ask that? Because the osteomyelites that are produced by trauma are initiated by a chill and the patient is incapacitated within the first twenty-four hours. Go on.

STUDENT: After you received the trauma, did you go to bed?

PATIENT: I was able to work until June 23d.

STUDENT: Why did you have to quit work then?

DR. MURPHY: The pain was so severe he could not work.

STUDENT: How soon after you quit work did you have to go to bed?

PATIENT: I did not go to bed until I came to the hospital.

DR. MURPHY: Was he able to walk without the aid of a cane or a crutch?

PATIENT: I could limp.

DR. MURPHY: Did you use a crutch?

PATIENT: No.

DR. MURPHY: Did you use a cane?

PATIENT: Sometimes.

DR. MURPHY: But it gave you pain all the time. Go on, what next?

STUDENT: Did it pain more at any time?

PATIENT: If I started to run.

STUDENT: Can you bend your leg? Can you turn it around in all directions?

PATIENT: When I attempt to turn around then it hurts.

STUDENT: When did you come to the hospital?

PATIENT: The sixteenth of July.

DR. MURPHY: Let us see what the skiagram showed on the sixteenth of July. What do you notice about the shaft of that femur?

STUDENT: It is thin.

DR. MURPHY: What else do you notice? The absence of what? The absence of compact bony tissue in the upper end of the femur. Notice that that looks like soft tissue in that neighborhood. It is not compact bony tissue. Let us follow the pictures. In the next the vascularization of the femur is still higher up and so on until finally it shows. Here it is still higher up. There it is enormously changed. It is almost a uniform destruction of the bone. The compact bony tissue is eaten out above and below. The question is, did he have a fracture originally? No. Has he had a fracture since? Can you put your weight on that leg?

PATIENT: No.

DR. MURPHY: What is the matter with him?

STUDENT: There are pathologic changes in the bone.

DR. MURPHY: That sounds good, but does not mean anything. Do not use words to cover up your lack of knowledge. What is the matter with him?

STUDENT: At the time of the accident he probably injured the blood-supply to the hip.

DR. MURPHY: Now that is just a dream. What is the matter with him?

STUDENT: He has an osteomalacia.

DR. MURPHY: That is another statement that does not mean anything. There has been an absorption of the bone, and disappearance of the compact bony tissue. There is an increased vascularization there. Is not that right? The bony tissue has changed. It resembles more closely the soft structures. In the other picture there is an effort at the reproduction of bone. Can you see it in the shaft? The outline of the bone is completely destroyed. What is that dense shadow in the center? What

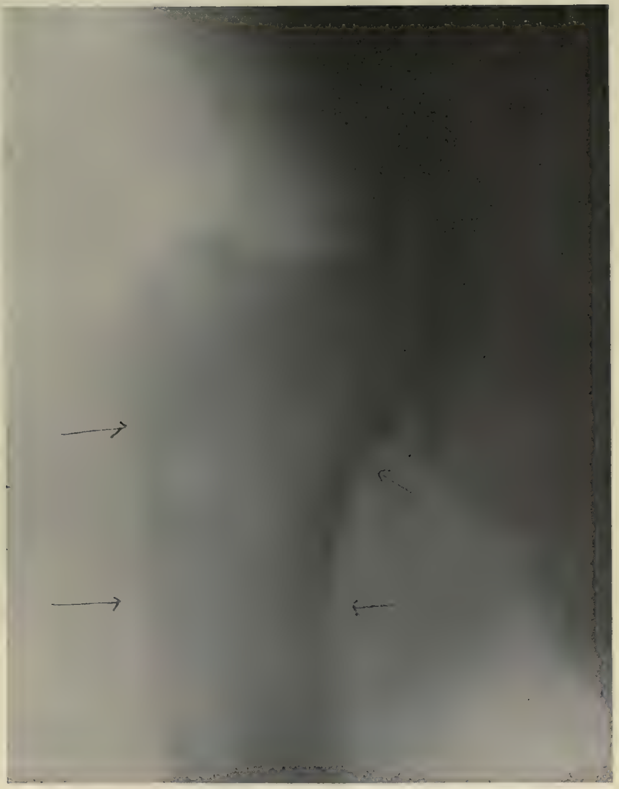

Fig. 259.—Traumatic sarcoma of femur. This skiagram was made about seven months after the injury. The disease has involved the upper extremity of the femur, except the greater trochanter, head and neck, and is beginning to extend out into the soft parts.

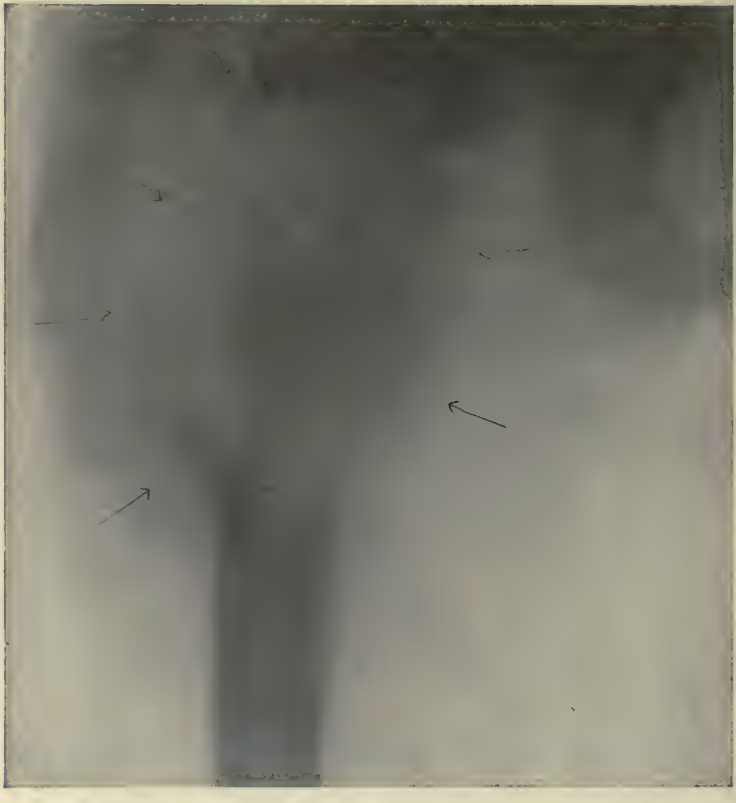

Fig. 260.—Traumatic sarcoma of femur. Skiagram made about nine months after the injury, showing the rapidity of growth of the tumor, which now extends outward into the soft parts on all sides, with destruction of all normal bone in the part of the femur involved. The great trochanter, head and neck of the bone, are now in the stage of the disease that the shaft was in two months previously, as shown in the preceding skiagram.

gives a shadow as dark as bone? Blood. An aneurysm will give a picture very much like bone. He has developed a large hematoma or an aneurysmal sarcoma in the center of that bone. Where there was first rarefaction, there is a deeper shadow. That deeper shadow is due to the fact that the tissue has now become a bloody mass in place of a cartilaginous mass. Often in these cases you have it looking exactly like bone.

The conclusion is then that there is a fracture as a result of a traumatic sarcoma.

He had no chill, no elevation of temperature, no leukocytosis. With the aneurysmal variety of sarcoma the patient may have a chill, elevation of temperature, and then a very rapid destruction of bone, which this man did not have. His hip became more and more painful as the compact bony tissue became absorbed, but the process did not reach the stage where the bone would have fractured voluntarily. If he kept on his feet, finally it would have fractured without the trauma. The shadow in the skiagram is that of an aneurysmal sarcoma. It looks like a bony neoplasm, but it is not.

Now then, as our time of adjournment is near, what is your conclusion?

STUDENT: It is a traumatic aneurysm.

DR. MURPHY: No, he has a traumatic sarcoma. There is no evidence that he has an aneurysm. That aneurysmal condition is due to the excess of blood that is under tension in the neoplasm; that he is about to have a fracture. He had an exploratory operation when he came in, but nothing was done. There is much granulation tissue there as the result of the operation, but the clinical history makes the diagnosis and the skiagram confirms it.

We have had here today two typical and classic cases, one of sarcoma, the sequence of a trauma, which should be diagnosed on the first skiagram and then treated either by an excision of the entire upper end of the femur with a transplant from the crest of the other tibia to take its place, or else treated by an exarticulation of the hip. The other case was a metastatic carcinoma of the femur which permits of no operation with the hope of a cure. That woman will live just as long without an operation. She is very thin, showing why the secondary neoplasm grew slowly, as the first one did. This second patient has no metastases in the lung, but probably there are metastases in other portions of his body. It is merely a question whether he should have the limb disarticulated at the hip-joint or whether he should be permitted to go just as he is. It is not good surgery to operate at all in those tissues, so we will give this man what little opportunity he may have to live for a while longer. He has an open wound infection, which in itself increases the hazard to his life immediately following the operation. An infection increases the hazard to life from a metastatic standpoint, because once you disturb them, the tumor cells are much more likely to be carried to some other portion of the body. [These cases may seem unnecessarily drawn out for the practitioner, but in teaching it is essential to keep the students thinking on the lines leading to a diagnosis based on the clinical history and the pathologic picture present. This is an illustrative clinic which will not be carried out in subsequent articles.—Ed.]

List of Cases Operated on and Demonstrated by Dr. John B. Murphy at Mercy Hospital During the Week of the Clinical Congress of Surgeons of North America

November 10–15, 1913

At the Clinics of Dr. Murphy at Mercy Hospital during the week of the Congress the following list of cases, for operation and demonstration, was handed to each of the doctors present. These will be published with comments, etc., in the coming numbers of the CLINICS under the same headings and numbers.

Case No.		
1.	**Room 118.**	Metastatic arthritis—Cured by vaccine—recurred.
2.	**Ward A.**	Compound fracture of fingers, with infection.
3.	**Room 132.**	Scarlatinal metastatic arthritis of hip, with rupture into rectum; associated with case of Jeffries, acute traumatic synovitis of hip, with rupture into rectum. See *x*-rays of both cases.
4.	**Room 144.**	Ankylosis of knee, following wiring of patella. Arthroplasty three weeks ago. Demonstration.
5.	**Room 138.**	Gastric ulcer, lesser curvature near pylorus. Posterior gastro-enterostomy; oblong button; demonstration; operated October 25.
6.	**Ward A.**	Pott's fracture; both malleoli fractured above level of articular tibial surface; retention by exarticular nailing, with demonstration of exarticular nailing for fixation of fracture near joint. For operation.
7.	**Room 138.**	Exostosis of elbow; operated October 28, 1913. Volkmann's contracture to be operated. (See also No. 110.)
8.	**Room 415.**	Ankylosis of jaw and hips; arthroplasty of jaw for operation.
9.	**Room 117.**	Trochanteric bursitis and metastatic osteochondritis of rib; operated October 26, 1913.
10.	**Room 136.**	Duodenal ulcer; posterior gastro-enterostomy; oblong button; with almost complete occlusion of the pylorus; operated October 30.
11.	**Room 138.**	Ankylosis of knee from penetrating wound. For operation—arthroplasty.
12.	**Ward B.**	Ununited fracture of the femur; Lane plate; for operation Thursday by Sir Arbuthnot Lane of London.
13.	**Ward B.**	Ununited fracture of neck of femur, showing progress of repair; operated nine weeks ago.
14.	**Room 201.**	Multiple arthritis; combined vaccine and injection treatment.
15.	**Ward B.**	Old compound infected fracture of the ankle.

Case No.	
16. Ward B.	Aneurysm of the aorta; spirochetic; by rest, salvarsan and mercury; compare x-rays.
17. Ward B.	Compound fracture of tibia and fibula; illustrating conservative treatment.
18.	(Mr. R.) Compound fracture of tibia and fibula with second operation.
19. Ward B.	Tuberculosis of the spine with paralysis; laminectomy.
20. Room 249.	Tuberculosis of the external portion of capsule of knee and external tuberosity; circumscribed; excision of diseased area; demonstration.
21. Room 249.	Metastatic arthritis of both knees; treated by injection and extension.
22. Ward B.	Ununited fracture of neck of femur; for operation—fixation.
23. Room 245.	Small round gastric ulcer; lesser curvature.
24. Room 212.	Carcinoma of the breast. Operated October 30; showing result of pectoral muscle axillary implantation. (See also 105.)
25. 226.	Branchial cyst, communicating with trachea.
26. Ward B.	Fracture of femur; plating; non-union; removal of plate.
27. Ward B.	Duodenal ulcer; previous perforation with drainage; for operation.
28. Ward B.	Charcot ankle; for arthrodesis.
29.	(Mr. V.) Double Charcot shoulder; for demonstration.
30. Room 258.	Dislocation of both semilunar cartilages (internal) with lax internal capsular ligament; for operation.
31. Room 230.	Multiple arthritis.
32. Room 355.	Carcinoma of rectum; for operation or demonstration.
33. Room 317.	Ununited fracture of lower end of humerus; transplantation from tibia.
34. Room 340.	Ununited fracture of femur; double plating; union.
35. Room 346.	Arthritis of both knees; hypertrophic synovitis with chronic hydrops.
36. Room 301.	Short circuit of large bowel without closing subileac loop; gas retention.
37. Room 353.	Compound fracture of tibia and fibula; transplantation of large flap from opposite limb; grafting.
38. Room 321.	Transplantation of pedicled flap for restoration of tendons (Dr. Golden).
39. Room 302.	Fracture of lower end of femur; plating; non-union; deformity; removal of plate; transplantation of bone; primary union.
40. Room 350.	Multiple arthritis from pharyngitis.
41. Room 330.	Cholecystitis without stone. Dr. Gaunt's case—septic cholecystitis.
42. Room 332.	Large carcinomatous prostate; enucleation; suprapubic.
43. Ward C.	Aneurysmal sarcoma of femur; primary.

Case No.		
44.	**Ward D.**	Metastatic carcinoma of femur five years after amputation of breast (Talbot). Demonstrate Mensenkampf *x*-rays.
45.	**Room 349.**	Sarcoma of the femur resembling tuberculosis; hip amputation.
46.	**Room 307.**	Ankylosis of hip; arthroplasty; aspiration of hematoma.
47.	**Room 344.**	Double congenital luxation of patella; one already operated, the other to be operated.
48.	**Room 348.**	Acquired luxation of patella; imbrication operation.
49.	**Room 338.**	Recurrent appendical abscess.
50.	**Room 342.**	Large ventral hernia; lateral imbrication operation.
51.	**Room 310.**	Gangrenous appendix.
52.	**Room 357.**	Ununited fracture of neck of femur, showing effect of traction through *x*-ray; for operation.
53.	**Room 304.**	Cholelithiasis; septic cholecystitis; resembling malaria.
54.	**Room 305.**	Tuberculous testicle; orchectomy; artificial implantation.
55.	**Room 308.**	Exophthalmic goiter with later development of myxedema, without operation.
56.	**Room 319.**	Exophthalmic goiter.
57.	**Room 328.**	Secondary carcinoma of upper lip five years after removal of carcinoma of lower lip.
58.	**Room 342.**	Multiple arthritis.
59.	**Room 322.**	Osteomyelitis of lower end of femur; secondary arthritis of knee.
60.	**Room 326.**	Fracture of both bones of forearm; malunion; refractured; medullary transplantation from tibia.
61.	**Room 318.**	Amputation neuritis; intradural division of cauda.
62.	**Room 354.**	Carcinoma of the tongue. Demonstrate also case of Brendt—tuberculosis of tongue; and Staneck—also tuberculosis of tongue. Show slides for last two cases. Also Conadley—tuberculosis of lower lip.
63.	**Room 316.**	Tuberculosis of tendon sheaths of dorsum of foot.
64.	**Room 334.**	Fracture of nose with depression.
65.	**Room 352.**	Metastatic arthritis of both knees from alveolar infection. Also demonstrate Mrs. Cox.
66.	**Room 428.**	Ununited fracture of femur; auto-transplantation from tibia.
67.	**Room 455.**	Tuberculosis of both hips with sinuses; auto-vaccines; demonstration of management.
68.	**Room 357.**	Ankylosis of hip; arthroplasty.
69.	**Room 455.**	Multiple arthritis from furunculosis.
70.	**Room 455.**	Tuberculosis of the knee; treated by injection, extension and tuberculin; demonstrate result.
71.	**Room 455.**	Metastatic infection of hip-joint with caries of bone; acute.
72.	**Room 448.**	Tuberculosis of condyle of femur with burrowing abscess; mixed infection.
73.	**Room 411.**	Abdominal adhesions; chronic cholecystitis.
74.	**Room 414.**	Acute intestinal obstruction by band.

Case No.		
75.	Room 446.	Sarcoma of lymph-glands of neck; treated with homo-Hodgkin's vaccine.
76.	Room 430.	Talipes equinovarus; excision of head of astragalus and tendon transplantation.
77.	Room 422.	Tubal pregnancy; ruptured in fourth month; live fetus.
78.	Room 446.	Ununited fracture of neck of femur; for operation.
79.	Room 406.	Large colloid goiter.
80.	Room 444.	Subhepatic gangrenous appendix.
81.	Room 412.	Uterine fibroid; hysterectomy.
82.	Room 347.	Double oöphorectomy; ventral implantation of fundus.
83.	Room 432.	Ununited fracture of neck of femur; for operation.
84.	Room 407.	Multiple arthritis; for injection and vaccines.
85.	Ward D.	Tuberculous knee; extension and tuberculin.
86.	Ward D.	Ununited fracture of neck of femur near the trochanter.
87.	Ward D.	Fracture of head of humerus; nailed.
88.	Ward D.	Carcinoma of uterus; zinc chlorid cauterization; hysterectomy.
89.	Ward D.	Carcinoma of lower end of esophagus.
90.	Ward D.	Transplantation of ovary.
91.	Ward D.	Sarcoma of radius.
92.		Bullet wound of pancreas.
93.		(Mr. M., Syracuse, N. Y.) Arthroplasty of elbow two years ago; ankylosis of wrist; for operation.
94.		(Dr. T.'s patient, Brunswick, Mo.) Exophthalmic goiter.
95.		(Mrs. X., Dubuque, Ia.) Carcinoma of breast.
96.		(Mr. W. R., Baribou.) Sarcoma of ulna.
97.		(Rev. H. W. B.) Fracture of skull; depression; epileptoid state; for operation.
98.	Room 324.	Carcinoma of stomach; for operation.
99.		(Mr. Nat G., Texas.) Hypertrophic tuberculous synovitis; excision; for operation.
100.		(Summers.) Carcinoma of superior maxillary bone; excision; for operation.
101.		(Mrs. L——al.) Ankylosis of knee; arthroplasty.
102.		(Master Geo. S., St. Louis.) Congenital aplasia; both bones of lower limb; fractures; double transplantation; union.
103.		(Mr. H., San Antonio.) Compound fracture of head of femur; removal; transplantation; ankylosis; arthroplasty.
104.		(Dr. C——ll.) Gun-shot wound of arm; fracture; musculospiral paralysis ; ankylosis of elbow; pseudo-arthritis; substitution for elbow-joint; transplantation of flexor carpi radialis to extensor carpi radialis and extensor longus pollicis; for operation.
105.		(Mrs. N——n.) Carcinoma of breast; operated thirteen

Case No.	
	years and eleven years with recurrence now; enormous edema of arm and face.
106.	(Mr. B——z, Sharon, Pa.) Fracture of neck of femur; non-union; late operation; repair.
107.	(Mr. McN——y.) Schlatter's disease, with elevation of patella.
108.	(Mr. W. D.) Ununited fracture of neck of femur with necrosis of head; restoration; union; regeneration; demonstration five years after operation. Also show *x*-rays of McDonald and Stearns. Also demonstrate four cases of reimplantation of luxated fractured head of humerus. Also Mrs. Gary, luxated head of humerus without fracture.
109.	(Mrs. Larson B.) Ankylosis of knee; arthroplasty; demonstration of result three years after operation.
110.	(Mr. W——t.) Volkmann's contracture; operated two years ago; demonstration of result with case No. 7.
111.	(Mr. ————.) Epithelioma of nose; thorium paste treatment; result.
112.	(Mrs. McE.) Hypertrophic lupus of nose; thorium paste; result.
113.	(Patient from Elgin.) Ankylosis of jaw; for operation.
114.	(Mr. W——ch.) Cholelithiasis; septic cholecystitis; for operation.
115.	(Mr. Sch——l.) Fracture of lower end of humerus; non-union; bone transplantation; refractured; secondary union of transplant only, with splendid functional result.
116.	(Mr. N——ge.) Sarcoma of trochanter of femur; exsection; transplantation from tibia, showing ossification and clinical result.
117.	(Miss Sc——l.) Bony ankylosis of elbow; arthroplasty; functional result.
118.	(Miss McA——.) Stenosis of pylorus; excision and gastroenterostomy.
119.	Anterior luxation of radius of elbow.
120.	Volkmann's contracture.
121.	Ankylosis of knee following cutting injury in region of knee-joint.
122.	Cyst of isthmus of thyroid gland.
123.	Cystadenoma of thyroid gland—developed during puberty—involvement of both lobes.
124-6.	Tuberculosis of lung. Pneumothorax treatment.
127.	Cystadenoma of right lobe of thyroid gland—developed during adult life.
128.	(Mrs. H.) Primary exophthalmic goiter; secondary colloid.
129.	(Mr. M. C. D.) Intestinal fistula; obstruction to common or hepatic duct; probably cholecystic. (With No. 53.)

Case No.		
130.		(Mr. N.) Electric cable destruction of the left arm.
131.		(Sister V.) Exophthalmic goiter. Early stage.
132.		(Mrs. D.) Tumor of brain, pontine angle. Operation Tuesday. Attendance by special invitation only.
133.		(Miss M.) Ununited fracture of both bones of the forearm; portion of radius absent. Transplantation of bone from tibia.
134.		(Mrs. X., Memphis.) Ankylosis of ankle (Dr. J.).
135.		(Mrs. N.) Bony ankylosis of knee; arthroplasty.
136.		(Mr. B.) Cholelithiasis with sepsis.
137.	**Ward A.**	Exophthalmic goiter.
138.	**Ward B.**	Tubercular knee.
139.	**Ward B.**	Perforated duodenal ulcer; admitted forty-eight hours after perforation.
140.	**Room 232.**	Injection of ilio-inguinal nerve.
141.	**Room 357.**	Cyst of breast.
142.	**Room 327.**	Abscess of pancreas.
143.	**Room 446.**	Multiple arthritis.
144.	**Room 409.**	Goiter.
145.	**Room 444.**	Umbilical hernia.
146.	**Room 446.**	Exploratory operation on kidney.
147.	**Ward D.**	Fracture of lower end of humerus.
148.	**Ward D.**	Pulmonary tuberculosis—nitrogen injection.
149.	**Ward D.**	Pulmonary tuberculosis—nitrogen injection.

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